```
=> d que stat l21
L1             1 SEA FILE=REGISTRY ABB=ON  BORON/CN
L2        192270 SEA FILE=HCAPLUS ABB=ON  (B OR T OR ?MYELOID? OR ?MAST? OR
                 ?PLASMA?)(W)?CELL?
L3         43423 SEA FILE=HCAPLUS ABB=ON  L2 AND (?DISEASE? OR ?DISORDER?)
L4           563 SEA FILE=HCAPLUS ABB=ON  L3 AND ((?DOMESTIC? OR ?COMPANION?)(W)
                 ?ANIMAL? OR DOG? OR CAT OR ?HORSE?)
L5             5 SEA FILE=HCAPLUS ABB=ON  L4 AND (?ANTIBOD?(2A)(?NAKED? OR
                 ?IMMUNOCONJUGAT? OR ?FUSION?(W)?PROTEIN? OR ?MULTISPECIFIC?))
L6           430 SEA FILE=HCAPLUS ABB=ON  L4 AND (?RADIOLABEL? OR ?CYTOKIN? OR
                 ?DRUG? OR ?TOXIN? OR ?RNASE? OR ?NEUTRON?(W)?CAPTUR?(W)(L1 OR
                 BORON)(W)ADD?) OR ?PHOTOACTIV?(W)(?AGENT? OR DYE?)
L7            42 SEA FILE=HCAPLUS ABB=ON  L6 AND (?MALIGN?(2A)((B OR T)(W)?CELL?
                 ) OR ?AUTOIMMUN?(W)(?DISEAS? OR ?DISORD?))
L8            15 SEA FILE=HCAPLUS ABB=ON  L7 AND ?BIND?
L9            10 SEA FILE=HCAPLUS ABB=ON  L7 AND (?CHEMOTHER?(W)(?AGENT? OR
                 ?DRUG?) OR ?IMMUNOMODULAT?)
L10            1 SEA FILE=HCAPLUS ABB=ON  L7 AND (?LABEL?(3A)?RADIONUCLID?)
L11            2 SEA FILE=HCAPLUS ABB=ON  L7 AND (?DIAGNOS?(W)(?AGENT? OR
                 ?COMPOSITION?))
L18           16 SEA FILE=HCAPLUS ABB=ON  L7 AND ((?RADIATION? OR ?CYTOKINE?)(W)
                 ?THERAP? OR ?IMMUNOSUPPRES?)
L19           35 SEA FILE=HCAPLUS ABB=ON  L5 OR L8 OR L9 OR L10 OR L11 OR L18
L21           25 SEA FILE=HCAPLUS ABB=ON  L19 AND (?METHOD? OR ?TECHNIQ? OR
                 ?PROCED?)

=> d ibib abs hitstr l21 1-25

L21  ANSWER 1 OF 25  HCAPLUS  COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER:         2003:875021  HCAPLUS
TITLE:                    Immunoglobulin fusion proteins as
                          bispecific antibodies and their use in
                          stimulating T-cell responses
INVENTOR(S):              Ren-Heidenreich, Lifen
PATENT ASSIGNEE(S):       Roger Williams Hospital, USA
SOURCE:                   PCT Int. Appl., 130 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE           APPLICATION NO.   DATE
     ---------------   ----  --------       ---------------   --------
     WO 2003090513      A2   20031106       WO 2003-US12772   20030423
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT,
             TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ,
             MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC,
             NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ,
             GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.:                  US 2002-374930P  P  20020423
AB   Bispecific antibodies in which the two antigen binding domains are
     incorporated into a fusion protein in which they are linked by a flexible
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linker peptide are described. One component is specific for a **disease** marker such as a tumor antigen and the other is specific for a **T cell** surface protein that will stimulate the **T cell**, especially CD3 antigens. These fusion proteins may be used to treat **disease** such as cancer by inducing a broad-based **T cell** recruitment. They avoid the adverse immune responses associated with mouse bispecific antibodies by limiting the fusion protein to using the antigen binding regions of the antibodies. This invention further provides related **methods** of treating a subject afflicted with a **disorder** mediated by the presence of an abnormal cell, and kits for practicing same. Construction of expression vectors for manufacture of a bispecific antibody to CD3 and the tumor antigen Ep-CAM is described. A combination of the antibody and interleukin 2 was able to prevent growth of LS174T tumors in SCID-Beige mice and even induce some necrosis.

L21 ANSWER 2 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:855818 HCAPLUS
TITLE: Treating an **autoimmune disease** using a soluble CTLA4 molecule in combination with a DMARD or NSAID **drug**
INVENTOR(S): Cohen, Robert; Carr, Suzette; Hagerty, David; Peach, Robert J.; Becker, Jean-Claude
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 339 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003088991	A1	20031030	WO 2003-US12356	20030418

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2002-373852P P 20020419
US 2002-407246P P 20020830

AB The present invention relates to compns. and **methods** for treating immune system **diseases** such as rheumatic **disease**, by administering to a subject soluble CTLA4 mols. that block endogenous B7 mols. from **binding** their ligands, alone, or in conjunction with other agents including **disease** modifying anti-rheumatic **drugs** (DMARDs) or non-steroidal anti-inflammatory **drugs** (NSAIDs). The soluble CTLA4 mol. comprises the extracellular domain (residues 1-124) of full-length human CTLA4, which may be fused at the N-terminus with the signal peptide of oncostatin M and at the C-terminal end with an Ig C γ1 domain. Single-site and double-site CTLA4 mutant sequences are also constructed, including L104E/A29Y-CTLA4/Ig, L104E/A29L-CTLA4/Ig, L104E/A29T-CTLA4/Ig, and L104E/A29W-CTLA4/Ig. CTLA4/Ig administered at 10 mg/kg (plus

methotrexate) has superior efficacy in treatment of rheumatoid arthritis compared to placebo (plus metrotrexate) based on efficacy parameters of the American Collage of Rheumatol. Core Data Set and Response Definitions (ACR). **Binding** kinetics to CD86 and CD80, pharmacokinetics, and pharmacodynamics of C-reactive protein, rheumatoid factor, interleukin-2 receptor, interleukin -6, and tumor necrosis factor α are provided.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 3 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:717505 HCAPLUS
DOCUMENT NUMBER: 139:244712
TITLE: Antibody compositions specific to p33QIK and p63krs1 polypeptides for diagnosis, prognosis and treatment of cell proliferation **diseases** or cancers
INVENTOR(S): Wang, Hwa-Chain Robert
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 75 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003170252	A1	20030911	US 2001-822110	20010330

PRIORITY APPLN. INFO.: US 2000-193550P P 20000331

AB Disclosed are compns. and **methods** for the detection and quantitation of mammalian p33QIK and p63Krs1 polypeptides in a sample. Also disclosed are **methods**, kits, and reagents for the diagnosis of **diseases** manifesting in or resulting from altered p33QIK and/or p63Krs1l polypeptide levels in a patient.

L21 ANSWER 4 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:656808 HCAPLUS
DOCUMENT NUMBER: 139:196278
TITLE: Anti-CD20 **antibodies** and **fusion proteins** for diagnosis and treatment of **B cell disease, B cell malignancy** and **autoimmune diseases**
INVENTOR(S): Hansen, Hans; Qu, Zhengxing; Goldenberg, David M.
PATENT ASSIGNEE(S): Immunomedics, Inc., USA; McCall, John Douglas
SOURCE: PCT Int. Appl., 106 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003068821	A2	20030821	WO 2003-GB665	20030214

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,

TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2002-356132P P 20020214
US 2002-416232P P 20021007

AB The present invention provides humanized, chimeric and human anti-CD20 antibodies and CD20 **antibody fusion proteins** that **bind** to a human **B cell** marker, referred to as CD20, which is useful for the treatment and diagnosis of **B -cell disorders**, such as **B-cell malignancies** and **autoimmune diseases**, and **methods** of treatment and diagnosis.

L21 ANSWER 5 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:570549 HCAPLUS
DOCUMENT NUMBER: 139:132441
TITLE: Co-stimulatory molecules derived from B7-1 antigens by recursive recombination with the ability to preferentially **bind** CD28 or CTLA-4 and to modulate **T cell** proliferation
INVENTOR(S): Punnonen, Juha; Lazetic, Alexandra; Leong, Steven R.; Chang, Chia-Chun; Apt, Doris; Gustafsson, Claes
PATENT ASSIGNEE(S): Maxygen, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 206 pp., Cont.-in-part of U.S. Ser. No. 888,324.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003138881	A1	20030724	US 2001-32214	20011220
WO 2002000717	A2	20020103	WO 2001-US19973	20010622
WO 2002000717	C2	20030206		
WO 2002000717	A3	20030821		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2003190697	A1	20031009	US 2001-888324	20010622

PRIORITY APPLN. INFO.: US 2000-213946P P 20000623
US 2000-241245P P 20001017
US 2001-888324 A2 20010622
WO 2001-US19973 A2 20010622

AB The invention provides polynucleotides and polypeptides encoded therefrom having advantageous properties, including an ability of the polypeptides to preferentially **bind** a CD28 or CTLA-4 receptor at a level greater or less than the ability of human B7-1 to **bind** CD28 or CTLA-4, or to induce or inhibit altered level of **T cell** proliferation response greater compared to that generated by human B7-1. The novel co-stimulatory mols. (NCSM) are derived by recursive

recombination of cDNAs encoding human, primate, cow, **cat**, and rabbit B7-1 to form libraries comprising two or more recombinant polynucleotides. Screening of the pooled recombined clones is based on preferential **binding** ability for soluble CD28 and CTLA-4 receptor fusion proteins. The polypeptides and polynucleotides of the invention are useful in therapeutic and prophylactic treatment **methods**, gene therapy applications, and vaccines.

L21 ANSWER 6 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:491393 HCAPLUS
DOCUMENT NUMBER: 139:51620
TITLE: Generation of hybrid cell lines to produce human monoclonal antibodies or fragments for diagnosis and therapy of infections and cancers
INVENTOR(S): Dessain, Scott; Weinberg, Robert
PATENT ASSIGNEE(S): Whitehead Institute for Biomedical Research, USA
SOURCE: PCT Int. Appl., 91 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003052082	A2	20030626	WO 2002-US40813	20021218

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.:
US 2001-341567P P 20011218
US 2002-349872P P 20020117
US 2002-355236P P 20020207
US 2002-375236P P 20020424

AB The present invention provides in one aspect novel fusion partner cells that ectopically express one or more genes that alter the phenotype of a hybrid cell made from a fusion of the fusion partner cell and a fusion cell, hybrid cell lines produced using the fusion partner cells. The invention in another aspect provides antibodies produced by certain hybrid cell lines, and compns. containing one or a combination of such antibodies or antigen-**binding** fragments thereof. The invention also provides in another aspect **methods** of using the antibodies or antigen-**binding** fragments thereof for diagnosis and treatment of **diseases** characterized by the antigens specifically bound by the antibodies.

L21 ANSWER 7 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:435384 HCAPLUS
DOCUMENT NUMBER: 139:21024
TITLE: CD24 and fusion proteins for treating **T cell**-mediated immune or autoimmune **diseases**
INVENTOR(S): Liu, Yang; Zheng, Pan; Bai, Xuefeng; Liu, Xingluo
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 34 pp., Cont.-in-part of U. S. Ser. No. 822,851.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003106084	A1	20030605	US 2002-119637	20020410
US 2003095966	A1	20030522	US 2001-822851	20010329

PRIORITY APPLN. INFO.: US 2000-192814P P 20000329
US 2001-822851 A2 20010329

AB **Methods** for blocking autoreactive **T cell** -initiated destruction of tissues in a mammal are provided. In one embodiment, the **method** comprises administering a purified CD24 polypeptide, a fusion protein comprising such polypeptide, or a biol. active fragment of such polypeptide to a mammalian subject who is suspected of having or predisposed to having an autoimmune **disease**. In another embodiment, anti-CD24 antibody or anti-CD24 Fab fragments are administered to the subject. In another embodiment, the **method** comprises administering a CD24 antisense mol., an expression vector encoding a CD24 antisense mol., CD24 dsRNAi, or an expression vector encoding CD24 dsRNAi to the subject. The present invention also relates to isolated and purified CD24 fusion proteins employed in the present **methods** and to transgenic mice that express the human CD24 protein on their **T cells** and/or their vascular endothelial cells but do not express murine heat shock antigen on any cells.

L21 ANSWER 8 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:434308 HCAPLUS
DOCUMENT NUMBER: 139:35063
TITLE: CD83 gene products for manipulating **cytokine** levels and treating **autoimmune disease**, allergy, cancer and infection
INVENTOR(S): Ramsdell, Fred; Proll, Sean C.; Staehling-Hampton, Karen; Appelby, Mark W.; Martinez, Leon Fernando Garcia
PATENT ASSIGNEE(S): Celltech R & D, Inc., USA
SOURCE: PCT Int. Appl., 158 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003045318	A2	20030605	WO 2002-US37738	20021121

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EC, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,

PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
PRIORITY APPLN. INFO.: US 2001-331958P P 20011121
AB The invention provides **methods** for modulating **cytokine** levels, GM-CSF levels and the immune system using CD83 nucleic acids, CD83 polypeptides, anti-CD83 antibodies and factors that influence CD83 activity or expression. The invention also provides mice having a mutant CD83 gene and mice having a transgenic CD83 gene, which are useful for defining the role of CD83 in the immune system and for identifying compds. that can modulate CD83 and the immune system.

L21 ANSWER 9 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:173821 HCAPLUS
DOCUMENT NUMBER: 138:220378
TITLE: Gene Aiolos knock-out mouse as Systemic Lupus Erythematosus (SLE) model, cured in double Aiolos/OBF-1 knock out mice, and **autoimmune disease drug** screening **methods**
INVENTOR(S): Matthias, Patrick Daniel; Sun, Jian
PATENT ASSIGNEE(S): Novartis Forschungsstiftung, Zweigniederlassung Friedrich Miescher Institute for Biomedical Research, Switz.
SOURCE: PCT Int. Appl., 49 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003018836	A2	20030306	WO 2002-EP9365	20020821
WO 2003018836	A3	20031030		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: GB 2001-20441 A 20010822
AB Homozygouse knock-out mice lacking the Aiolos gene are shown to exhibit multiple phenotypes in common with humans suffering from the **autoimmune disease** Systemic Lupus Erythematosus (SLE). When Aiolos -/- mice are crossed with homozygous knock out mice lacking the OBF-1 transcription factor gene, resultant double knock out mice lack all signs of SLE. **Methods** of screening for agents active against **autoimmune diseases**, for example SLE are provided. In vitro **methods** include screening for antagonists of OBF-1, screening for agents which inhibit **binding** of OBF-1 to oct-1 or oct-2, screening for agonists or antagonists of Aiolos protein and screening for agents which upregulate expression of Aiolos or down-regulate expression of OBF-1. Also disclosed are **methods** of screening using knock-out mice and **B cells** from knock-out mice.

L21 ANSWER 10 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:906554 HCAPLUS
DOCUMENT NUMBER: 138:1044
TITLE: G protein-coupled receptor (GPCR) microarrays for determination of GPCR gene expression profiles and uses in **drug** and **toxin** screening and diagnostics
INVENTOR(S): Thirstrup, Kenneth; Madsen, Lars Siim; Jensen, Jens Bitsch; Hummel, Rene; Jensen, Bo Skaaning
PATENT ASSIGNEE(S): Azign Bioscience A/s, Den.
SOURCE: PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002095065	A2	20021128	WO 2002-DK337	20020521

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-802 A 20010518
AB The invention provides G protein-coupled receptor (GPCR) arrays, kits comprising GPCR arrays and **methods** to produce such GPCR arrays. GPCR arrays are useful in the determination of GPCR expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved in the response of the GPCR expression. The invention relates to an GPCR array comprising a multiplicity of individual GPCR polynucleotide spots stably associated with a surface of a solid support, wherein an individual GPCR polynucleotide spot comprises an GPCR polynucleotide composition comprising a non-conserved region of an GPCR polynucleotide family member, the spots representing at least two different regions of an GPCR polynucleotide member of a family. The invention also relates to a set of primers specific for nonconserved regions of GPCR polynucleotide family members, wherein the set of primers are used in the **method** for the production of an array according to the invention. In still a further aspect, the invention relates to a diagnostic **method** to determine the differences of GPCR expression profiles between two different biol. materials.

L21 ANSWER 11 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:906553 HCAPLUS
DOCUMENT NUMBER: 138:1043
TITLE: Transporter microarrays for the determination of transporter gene expression profiles and uses in **drug** and **toxin** screening and diagnostics
INVENTOR(S): Jensen, Jens Bitsch; Madsen, Lars Siim; Gether, Ulrik; Jensen, Bo Skaaning
PATENT ASSIGNEE(S): Azign Bioscience A/S, Den.

SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002095064	A1	20021128	WO 2002-DK336	20020521

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-803 A 20010518

AB The object of the invention is to provide transporter arrays, kits comprising transporter arrays and **methods** to produce such transporter arrays. Transporter arrays are useful in the determination of transporter expression profiles in biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the transporter expression. The invention relates to an transporter array comprising a multiplicity of individual transporter polynucleotide spots stably associated with a surface of a solid support, wherein an individual transporter polynucleotide spot comprises an transporter polynucleotide composition comprising a non-conserved region of an transporter polynucleotide family member, the spots representing at least two different regions of a transporter polynucleotide. A set of primers specific for nonconserved regions of transporter polynucleotide family members are provided, wherein the set of primers are used in the **method** for the production of an array according to the invention. A diagnostic **method** detecting the differences of transporter expression profiles between two different biol. materials is also provided.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 12 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:832908 HCAPLUS
DOCUMENT NUMBER: 137:347474
TITLE: Ion channel microarrays for the determination of ion channel gene expression profiles and uses in **drug** and **toxin** screening and diagnostics
INVENTOR(S): Jensen, Bo Skaaning; Madsen, Lars Siim; Jensen, Jens Bitsch; Kjaer, Katrine
PATENT ASSIGNEE(S): Neurosearch A/S, Den.
SOURCE: PCT Int. Appl., 53 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 2002086050	A2	20021031	WO 2002-DK253	20020418

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: DK 2001-635 A 20010420

AB The invention provides completely novel and improved ion channel arrays, kits comprising ion channel arrays and **methods** to produce such ion channel arrays. Ion channel arrays are useful in the determination of ion channel expression profiles in a certain biol. material, several biol. materials and also in the identification of therapeutic, prophylactic and/or toxic agents involved either directly or indirectly in the response of the ion channel expression. In a first aspect the invention relates to an ion channel array comprising a multiplicity of individual ion channel polynucleotide spots stably associated with a surface of a solid support, wherein an individual ion channel polynucleotide spot comprises an ion channel polynucleotide composition comprising a non-conserved region of an ion channel polynucleotide family member, the spots representing at least two different regions of an ion channel polynucleotide member of a family. In a further aspect, the invention relates to a set of primers specific for nonconserved regions of ion channel polynucleotide family members, wherein the set of primers are used in the **method** for the production of an array according to the invention. In still a further aspect, the invention relates to a diagnostic **method** to determine the differences of ion channel expression profiles between two different biol. materials; said **method** comprises obtaining a first ion channel expression profile of a first biol. material according to the **method** of the present invention, obtaining a second ion channel expression profile of a second biol. material according to the **method** of the present invention, comparing the first and second ion channel expression profiles, and identifying any difference in the ion channel expression profile.

L21 ANSWER 13 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:556017 HCAPLUS
DOCUMENT NUMBER: 137:124187
TITLE: Extracorporeal **methods** comprising photopheresis treatment for enhancing antigen presentation and immune responsiveness to T- or **B-cell malignancies** and **autoimmune diseases**
INVENTOR(S): Edelson, Richard L.
PATENT ASSIGNEE(S): Morgan, Lewis, Bockius Llp, USA
SOURCE: U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U. S. Ser. No. 621,109.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002098469	A1	20020725	US 1998-14705	19980128

WO 9734472	A1	19970925	WO 1997-US4285	19970318	
W: AU, CA, JP, MX, US, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
CA 2318991	AA	19990805	CA 1999-2318991	19990128	
WO 9938380	A1	19990805	WO 1999-US1729	19990128	
W: AU, CA, JP, US					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
AU 9924750	A1	19990816	AU 1999-24750	19990128	
EP 1054591	A1	20001128	EP 1999-904333	19990128	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
JP 2002501726	T2	20020122	JP 2000-529128	19990128	
PRIORITY APPLN. INFO.:			US 1996-621109	A2 19960322	
			WO 1997-US4285	A2 19970318	
			US 1998-14705	A 19980128	
			WO 1999-US1729	W 19990128	

AB **Methods** are provided to enhance the expression on certain disease-effector cells of MHC peptides and/or the amount of disease-associated antigens presented by such MHC peptides. These **methods** relate to certain incubation times for such cells following photopheresis treatment and also relate to appropriate incubation containers. Overall, these **methods** enhance a subject's immune response to disease-associated antigens expressed, for example, by clonal T-cell or **B-cell malignancies** and in **T-cell** or B-cell mediated **autoimmune disorders.**

L21 ANSWER 14 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:923628 HCAPLUS
DOCUMENT NUMBER: 136:52724
TITLE: **Methods** for regulating a cell-mediated immune response by blocking lymphocytic signals and by blocking LFA-1 mediated adhesion
INVENTOR(S): Townsend, Robert M.; Todderud, Charles Gordon; Peach, Robert J.
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001095928	A2	20011220	WO 2001-US18619	20010608
WO 2001095928	A3	20020530		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002039577	A1	20020404	US 2001-877987	20010608
EP 1294391	A2	20030326	EP 2001-942118	20010608
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				

PRIORITY APPLN. INFO.: US 2000-210671P P 20000609
WO 2001-US18619 W 20010608

AB The invention provides **methods** for regulating cell-mediated immune responses, immune system **diseases** and allograft transplant rejection by interfering with the interaction of at least three different cell surface mols. with their natural ligands. A first cellular interaction is mediated by CD28/B7/CTLA4, a second interaction is mediated by CD40/CD154, and a third interaction is mediated by LFA-1 interaction with its ligands. Regulation of a cell-mediated immune response affects immune system **diseases** such as those associated with allograft transplantation.

L21 ANSWER 15 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2001:417021 HCAPLUS
DOCUMENT NUMBER: 135:45195
TITLE: Feline immunoregulatory proteins, nucleic acid molecules, and uses thereof
INVENTOR(S): Wonderling, Ramani S.
PATENT ASSIGNEE(S): Heska Corporation, USA
SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001040313	A2	20010607	WO 2000-US32826	20001201
WO 2001040313	A3	20011018		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6482403	B1	20021119	US 1999-451527	19991201
AU 2001019416	A5	20010612	AU 2001-19416	20001201

PRIORITY APPLN. INFO.:
US 1999-451527 A1 19991201
US 1998-87306P P 19980529
US 1999-322409 A2 19990528
WO 2000-US32826 W 20001201

AB The present invention relates to feline interferon alpha protein; to feline interferon alpha nucleic acid mols.; to antibodies raised against such proteins; and to inhibitory compds. that regulate such proteins. The present invention also includes **methods** to identify and obtain such proteins, nucleic acid mols., antibodies, and inhibitory compds. Also included in the present invention are therapeutic compns. comprising such proteins, nucleic acid mols., antibodies and/or inhibitory compds. as well as the use of such therapeutic compns. to regulate an immune response in an animal.

L21 ANSWER 16 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:756562 HCAPLUS
DOCUMENT NUMBER: 133:320984
TITLE: Differentiation of monocytes into functional dendritic cells

INVENTOR(S): Edelson, Richard Leslie
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000062818	A1	20001026	WO 2000-US8793	20000403
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1176986	A1	20020206	EP 2000-921611	20000403
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2001053355	A1	20011220	US 2001-928855	20010813
US 6607722	B2	20030819		
US 2002004044	A1	20020110	US 2001-928811	20010813
US 6524855	B2	20030225		

PRIORITY APPLN. INFO.: US 1999-294494 A 19990420
WO 2000-US8793 W 20000403

AB A **method** for inducing differentiation of monocytes contained in an extracorporeal quantity of a subject's blood into functional dendritic antigen presenting cells is provided. The monocytes are first treated by exposure to phys. perturbation, irradiation in the presence of a **photoactivatable agent** capable of forming photoadducts with cellular DNA components, and/or treatment with a DNA **binding** agent. The treated monocytes are then incubated for a period of time sufficient to maximize the number of functional dendritic cells in the treated cell population. Functional dendritic cells generated from induced monocytes are incubated together with disease effector agents to enhance the presentation of at least one disease-causing antigen expressed by the disease effector agents. Compns. including dendritic cells derived from induced monocytes and compns. including such dendritic cells incubated with disease effector agents are also provided.

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 17 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:725641 HCAPLUS
DOCUMENT NUMBER: 133:276336
TITLE: Monoesters of methylenediphosphonic acids and salts selectively inhibiting Tγ9δ2 lymphocytes
INVENTOR(S): Belmant, Christian; Bonneville, Marc; Peyrat, Marc Alix; Fournie, Jean-jacques; Kozikowski, Alan P.
PATENT ASSIGNEE(S): Institut National de la Sante et de la Recherche Medicale, Fr.; Sangstat Medical Corp.
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000059916	A1	20001012	WO 2000-FR837	20000404
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
FR 2791981	A1	20001013	FR 1999-4263	19990406
FR 2791981	B1	20010720		
EP 1165573	A1	20020102	EP 2000-917119	20000404
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 6624151	B1	20030923	US 2001-958323	20011220

PRIORITY APPLN. INFO.: FR 1999-4263 A 19990406
WO 2000-FR837 W 20000404

OTHER SOURCE(S): MARPAT 133:276336

AB The invention concerns compds. CH3-R1-(CH2)2-R2 wherein: R1 = CMe(OH), C(CH2OH)(OH), C(CH2X)(OH) (X = Cl, Br, I), C(OCH2) (epoxide), C(:CH2), C(C(O)H)R3 (R3 = H, OH); and R2 = OP(O)(OCat)CR42P(O)(OCat)2 (CR42 = CH2, CF2, CHF; **Cat** = organic cation or proton); an example is CH2:CMeCH2CH2OP(O)(ONBu4)CH2P(O)(ONBu4)2. The invention also concerns the uses of said compds. as selective inhibitors of Tγ9δ2 lymphocytes, and their uses, in particular for therapeutic purposes. Although the **method** of preparation is not claimed, the prepns. of 9 compds. are given in the examples section.

REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 18 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:513450 HCAPLUS
DOCUMENT NUMBER: 133:134158
TITLE: Activation of regulatory **T cells** by alpha-melanocyte stimulating hormone
INVENTOR(S): Taylor, Andrew W.; Nishida, Tomomi
PATENT ASSIGNEE(S): Schepens Eye Research Institute, Inc., USA
SOURCE: PCT Int. Appl., 111 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000042856	A1	20000727	WO 2000-US1608	20000121
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2359636	AA	20000727	CA 2000-2359636	20000121
EP 1150570	A1	20011107	EP 2000-909957	20000121
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

JP 2002534968 T2 20021022 JP 2000-594330 20000121
US 2002090724 A1 20020711 US 2001-912670 20010723
PRIORITY APPLN. INFO.: US 1999-116851P P 19990122
US 1999-156788P P 19990930
WO 2000-US1608 W 20000121

AB The invention encompasses a **method** of down-regulating a **T cell**-mediated immune response, through activation or **T cell** receptor (TCR) stimulation of antigen-primed **T cells** in the presence of alpha-MSH (α-MSH), which may be optionally enhanced by adding transforming growth factor-β2 (TGF-β2) approx. 4-6 h after the start of the primed **T cells'** exposure to α-MSH. Activation of the primed **T cells** may be mediated by presentation of the specific antigen to the primed **T cells**, or by an anti-TCR antibody or a **T cell** mitogen. As a result of the α-MSH treatment modulating the **T cell** activation, antigen-specific, regulatory, CD4+/CD25+ **T cells** are generated that produce transforming growth factor-β (TGF-β) and can non-specifically down-regulate Th1-mediated inflammatory activities. The **method** may be used to down-regulate or suppress an autoimmune condition or a graft rejection in a transplant patient. The invention also encompasses a kit for generating regulatory **T cell** comprising a specific antigen, α-MSH, and optionally, TGF-β2 and/or a **T cell** culture medium. Also provided are gene therapy treatments for suppressing an autoimmune or graft rejection response, or for re-establishing auto-tolerance, by introducing genetic material (e.g. nucleic acid) for expressing α-MSH or a receptor- **binding** portion thereof, into a localized tissue site.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 19 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2000:190949 HCAPLUS
DOCUMENT NUMBER: 132:246351
TITLE: **Method** of using zonula occludens **toxin** (Zot) or zonulin to inhibit lymphocyte proliferation in an antigen-specific manner
INVENTOR(S): Fasano, Alessio; Sztein, Marcelo B.; Lu, Ruiliang; Tanner, Michael K.
PATENT ASSIGNEE(S): University of Maryland, Baltimore, USA
SOURCE: PCT Int. Appl., 95 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000015252	A1	20000323	WO 1999-US18842	19990909

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2342771	AA	20000323	CA 1999-2342771	19990909
AU 9960190	A1	20000403	AU 1999-60190	19990909
AU 754142	B2	20021107		
EP 1113813	A1	20010711	EP 1999-969032	19990909
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002524531	T2	20020806	JP 2000-569836	19990909
NZ 510150	A	20021025	NZ 1999-510150	19990909
RU 2214271	C2	20031020	RU 2001-110101	19990909
NO 2001001253	A	20010315	NO 2001-1253	20010313
ZA 2001002071	A	20020225	ZA 2001-2071	20010313
PRIORITY APPLN. INFO.:			US 1998-100266P P	19980914
			WO 1999-US18842 W	19990909

AB **Methods** for using Zot or zonulin as an antigen-specific inhibitor of antigen-presenting cell (APC) activity and lymphocyte proliferation, being primarily useful in the field of immunoregulation and immunotherapy, are described. Specifically, Zot and zonulin inhibit antigen-presenting cell-mediated antigen-specific lymphocyte proliferation in a dose-dependent manner. This effect is associated with the presence of a macrophage surface receptor to which Zot **binds** in a specific and saturable way. This down-regulation of the immune response is, at least in part, associated with a decreased uptake of antigen.

REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 20 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:495127 HCAPLUS
DOCUMENT NUMBER: 131:129043
TITLE: Improved extracorporeal **methods** for enhancing antigen presentation and immune responsiveness
INVENTOR(S): Edelson, Richard L.
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9938380	A1	19990805	WO 1999-US1729	19990128
W: AU, CA, JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 2002098469	A1	20020725	US 1998-14705	19980128
CA 2318991	AA	19990805	CA 1999-2318991	19990128
AU 9924750	A1	19990816	AU 1999-24750	19990128
EP 1054591	A1	20001128	EP 1999-904333	19990128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002501726	T2	20020122	JP 2000-529128	19990128
PRIORITY APPLN. INFO.:			US 1998-14705 A	19980128
			US 1996-621109 A2	19960322
			WO 1997-US4285 A2	19970318
			WO 1999-US1729 W	19990128

AB **Methods** are provided to enhance the expression on certain disease-effector cells of MHC peptides and/or the amount of disease-associated antigens presented by such MHC peptides. These **methods** relate

to certain incubation times for such cells following photopheresis treatment and also relate to appropriate incubation containers. Overall, these **methods** enhance a subject's immune response to disease-associated antigens expressed, for example, by clonal T-cell or **B-cell malignancies** and in **T-cell** or B-cell mediated **autoimmune disorders**.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 21 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:404859 HCAPLUS
DOCUMENT NUMBER: 131:57772
TITLE: **Methods** to treat undesirable immune responses
INVENTOR(S): Conti-Fine, Bianca M.
PATENT ASSIGNEE(S): Regents of the University of Minnesota, USA
SOURCE: PCT Int. Appl., 221 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9930736	A2	19990624	WO 1998-US26787	19981216
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2315537	AA	19990624	CA 1998-2315537	19981216
AU 9931799	A1	19990705	AU 1999-31799	19981216
EP 1037663	A2	20000927	EP 1998-967008	19981216
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1997-991143 A2 19971216
WO 1998-US26787 W 19981216

AB Isolated and purified peptides and variants thereof, useful to prevent or treat antibody-mediated **diseases**, or indications caused by an undesirable antibody response to a given antigen, are provided. Also provided are peptides and **methods** useful to prevent or treat indications associated with the use of viral vectors in gene replacement therapy. Further, a **method** to inhibit or prevent aberrant immune responses to exogenous, non-infectious antigen is provided. The antigen associated with the antibody-mediated **disease** is an endogenous antigen such as acetylcholine receptor, insulin, growth hormone, factor VIII or factor IX; or an exogenous antigen such as fungal antigen, plant antigen, domestic **cat** antigen or mite allergen. The antibody-mediated **disease** is an **autoimmune disease**, allergic **disease**, systemic lupus erythematosus, pemphigus, thrombic thrombocytopenic purpura, hemophilia A, hemophilia B, or myasthenia gravis.

L21 ANSWER 22 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1997:684303 HCAPLUS

DOCUMENT NUMBER: 127:358050
TITLE: Novel product and process for T lymphocyte veto
INVENTOR(S): Staerz, Uwe D.
PATENT ASSIGNEE(S): National Jewish Center for Immunology and Respiratory Medicine, USA; Staerz, Uwe D.
SOURCE: PCT Int. Appl., 116 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9737687	A1	19971016	WO 1997-US5943	19970410
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6060054	A	20000509	US 1996-630172	19960410
CA 2251819	AA	19971016	CA 1997-2251819	19970410
AU 9727258	A1	19971029	AU 1997-27258	19970410
EP 929316	A1	19990721	EP 1997-921134	19970410
R: CH, DE, FR, GB, IT, LI, SE				
US 6264950	B1	20010724	US 1999-375419	19990817

PRIORITY APPLN. INFO.: US 1996-630172 A2 19960410
WO 1997-US5943 W 19970410

AB The present invention relates to a product and process for suppressing an immune response using a T lymphocyte veto mol. capable of blocking cell surface mols. responsible for **T cell** activation. Disclosed is a CD4 or CD2 mol., associated with an Ig mol. capable of **binding** to a major histocompatibility antigen. The CD2 or CD4 mol. may also be replaced by CTLA4, Fas ligand, CD5, CD7, CD9, CD11, CD18, CD27, CD43, CD45. CD48, B7.1 or B7.2 protein. Also disclosed is a **method** to produce a T lymphocyte veto mol., a therapeutic composition comprising a T lymphocyte veto mol. and **methods** to use T lymphocyte veto mols. in therapeutic processes requiring suppression of an immune response.

L21 ANSWER 23 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1997:326866 HCAPLUS
DOCUMENT NUMBER: 126:308798
TITLE: Chimeric DNA-**binding**/DNA methyltransferase nucleic acid and polypeptide and their uses
INVENTOR(S): Bestór, Timothy H.
PATENT ASSIGNEE(S): Trustees of Columbia University in the City of New York, USA; Bestor, Timothy H.
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

WO 9711972	A1	19970403	WO 1996-US15576	19960927	
W: AU, CA, JP, MX, US, US					
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
AU 9673781	A1	19970417	AU 1996-73781	19960927	
US 2002188103	A1	20021212	US 1998-51013	19981009	
PRIORITY APPLN. INFO.:			US 1995-4445P	P	19950928
			US 1996-594866	A2	19960131
			WO 1996-US15576	W	19960927

AB The present invention provides a chimeric protein which comprises a mutated DNA methyltransferase portion and a DNA **binding** protein portion that **binds** sufficiently close to a promoter sequence of a target gene (which promoter sequence contains a methylation site) to specifically methylate the site and inhibit activity of the promoter and thus inhibit expression of the target gene. This invention also provides for a **method** for inhibiting the expression of a target gene which includes contacting a promoter of the target gene with the chimeric protein, so as to specifically methylate the promoter sequence of the target gene thus inhibiting expression of the target gene.

L21 ANSWER 24 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1994:532198 HCAPLUS
DOCUMENT NUMBER: 121:132198
TITLE: Specific immune system modulation by forming specific antigen-presenting cells
INVENTOR(S): Edelson, Richard L.; Gasparro, Francis P.; Tigelaar, Robert E.
PATENT ASSIGNEE(S): Yale University, USA
SOURCE: PCT Int. Appl., 58 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9411016	A1	19940526	WO 1993-US11220	19931118	
W: JP, US					
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
EP 668772	A1	19950830	EP 1994-901598	19931118	
EP 668772	B1	20000830			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE					
JP 08503470	T2	19960416	JP 1993-512497	19931118	
AT 195875	E	20000915	AT 1994-901598	19931118	
ES 2149254	T3	20001101	ES 1994-901598	19931118	
US 6355238	B1	20020312	US 1997-822940	19970321	
PRIORITY APPLN. INFO.:			US 1992-977672	A	19921118
			WO 1993-US11220	W	19931118

AB **Methods** and pharmaceutical compns. for modifying the immune response of a mammal to a specific antigen are provided. The **methods** include treating an antigen presenting cell (leukocyte) with **photoactivatable agent** at 20-28° to enhance expression of a major histocompatibility complex mol. (class I or II) and reacting the treated antigen presenting cell with the antigen extracorporeally to form an antigen-associated antigen presenting cell. Thus, adducts of 8-methoxypsoralen and lysozyme, chick ovalbumin and a HIV inhibiting peptide were formed and characterized. Antigen-presenting cells were formed and used to paralyze immune response by clonal anergy or to induce an anti-idiotypic suppressor response for treating rheumatoid arthritis, and for preventing allograft rejection and allergic contact

dermatitis.

L21 ANSWER 25 OF 25 HCAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1989:495435 HCAPLUS
DOCUMENT NUMBER: 111:95435
TITLE: Active specific immune activation or suppression by photopheresis
INVENTOR(S): Edelson, Richard L.; Tripodi, Daniel J.
PATENT ASSIGNEE(S): Therakos, Inc., USA
SOURCE: Eur. Pat. Appl., 16 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 284409	A2	19880928	EP 1988-302660	19880325
EP 284409	A3	19890208		
R: AT, BE, DE, ES, FR, GB, IT, LU, NL, SE				
AU 8813584	A1	19880929	AU 1988-13584	19880324
AU 606666	B2	19910214		
DK 8801666	A	19880928	DK 1988-1666	19880325
JP 63275525	A2	19881114	JP 1988-69892	19880325
JP 2958372	B2	19991006		
ZA 8802159	A	19891129	ZA 1988-2159	19880325
CA 1306678	A1	19920825	CA 1988-562446	19880325

PRIORITY APPLN. INFO.: US 1987-31490 19870327

AB A **method** for specifically altering the immune system response of a mammal to a specific antigen comprises (a) contacting the mammal's immune system with the antigen to artificially stimulate the immune system; (b) withdrawing immune system cells (e.g. blood including antigen-stimulated cells) from the mammal; (c) treating the cells so as to alter the antigen-stimulated cells; and (d) returning the material and altered cells to the mammal. Preferably, the **method** involves photopheresis in which the cells are treated with a **photoactivatable agent**, the agent is activated with UV irradiation, and the treated cells are returned to the patient for immune system activation or suppression. Young (4-6 wk old) MRL/lpr mice, a strain which develops an **autoimmune disease** similar to human systemic lupus erythematosus, were treated, prior to onset of **autoimmune disease**, with 8-methoxypsoralen-UVA inactivated syngeneic splenocytes from old autoimmune mice (18-22 wk old). The mice received biweekly injections of 20 + 106-50 + 106 splenocytes treated with 100 ng 8-methoxypsoralen/mL and 1 J/cm2 UVA. Fulminant lymphoid hyperplasia was delayed in onset and treated mice survived >2 mo longer than untreated littermates. In addition, the production of anti-DNA autoantibodies was inhibited in the treated mice. The spleens contained fewer T-cells and increased percentages of B-cells and Ia+ cells. The treated mice retained the capacity to mount a proliferative response to T- and B-cell mitogens.

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=> d que stat l23
L1             1 SEA FILE=REGISTRY ABB=ON  BORON/CN
L2        192270 SEA FILE=HCAPLUS ABB=ON  (B OR T OR ?MYELOID? OR ?MAST? OR
                 ?PLASMA?)(W)?CELL?
L3         43423 SEA FILE=HCAPLUS ABB=ON  L2 AND (?DISEASE? OR ?DISORDER?)
L4           563 SEA FILE=HCAPLUS ABB=ON  L3 AND ((?DOMESTIC? OR ?COMPANION?)(W)
                 ?ANIMAL? OR DOG? OR CAT OR ?HORSE?)
L5             5 SEA FILE=HCAPLUS ABB=ON  L4 AND (?ANTIBOD?(2A)(?NAKED? OR
                 ?IMMUNOCONJUGAT? OR ?FUSION?(W)?PROTEIN? OR ?MULTISPECIFIC?))
L6           430 SEA FILE=HCAPLUS ABB=ON  L4 AND (?RADIOLABEL? OR ?CYTOKIN? OR
                 ?DRUG? OR ?TOXIN? OR ?RNASE? OR ?NEUTRON?(W)?CAPTUR?(W)(L1 OR
                 BORON)(W)ADD?) OR ?PHOTOACTIV?(W)(?AGENT? OR DYE?)
L7            42 SEA FILE=HCAPLUS ABB=ON  L6 AND (?MALIGN?(2A)((B OR T)(W)?CELL?
                 ) OR ?AUTOIMMUN?(W)(?DISEAS? OR ?DISORD?))
L8            15 SEA FILE=HCAPLUS ABB=ON  L7 AND ?BIND?
L9            10 SEA FILE=HCAPLUS ABB=ON  L7 AND (?CHEMOTHER?(W)(?AGENT? OR
                 ?DRUG?) OR ?IMMUNOMODULAT?)
L10            1 SEA FILE=HCAPLUS ABB=ON  L7 AND (?LABEL?(3A)?RADIONUCLID?)
L11            2 SEA FILE=HCAPLUS ABB=ON  L7 AND (?DIAGNOS?(W)(?AGENT? OR
                 ?COMPOSITION?))
L18           16 SEA FILE=HCAPLUS ABB=ON  L7 AND ((?RADIATION? OR ?CYTOKINE?)(W)
                 ?THERAP? OR ?IMMUNOSUPPRES?)
L19           35 SEA FILE=HCAPLUS ABB=ON  L5 OR L8 OR L9 OR L10 OR L11 OR L18
L21           25 SEA FILE=HCAPLUS ABB=ON  L19 AND (?METHOD? OR ?TECHNIQ? OR
                 ?PROCED?)
L22           29 SEA L21
L23           29 DUP REMOV L22 (0 DUPLICATES REMOVED)

=> d ibib abs l23 1-29

L23  ANSWER 1 OF 29  WPIDS  COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER:     2003-697522 [66]   WPIDS
DOC. NO. NON-CPI:     N2003-556970
DOC. NO. CPI:         C2003-191816
TITLE:                New humanized anti-CD20 monoclonal antibody (MAb) that
                      retains substantially the B-cell and
                      B-cell lymphoma and leukemia cell
                      targeting of the murine anti-CD20 MAb, useful for
                      treating B-cell lymphoma, leukemia or
                      an autoimmune diseases.
DERWENT CLASS:        B04 D16 K08 S03
INVENTOR(S):          GOLDENBERG, D M; HANSEN, H; QU, Z
PATENT ASSIGNEE(S):   (IMMU-N) IMMUNOMEDICS INC; (MCCA-I) MCCALL J D
COUNTRY COUNT:        101
PATENT INFORMATION:

     PATENT NO   KIND DATE     WEEK      LA   PG
     -----------------------------------------
     WO 2003068821 A2 20030821 (200366)* EN  106
        RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS
            LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
         W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK
            DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR
            KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT
            RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM
            ZW

APPLICATION DETAILS:
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PATENT NO	KIND	APPLICATION	DATE
WO 2003068821 A2		WO 2003-GB665	20030214

PRIORITY APPLN. INFO: US 2002-416232P 20021007; US 2002-356132P 20020214

AN 2003-697522 [66] WPIDS

AB WO2003068821 A UPAB: 20031014

NOVELTY - A humanized anti-CD20 (hCD20) monoclonal antibody (MAb) or its antigen-**binding** fragment comprising the complementarity determining regions (CDRs) of at least one murine anti-CD20 MAb variable region and the framework regions (FRs) of at least one human IVlAb variable region, where humanized anti-CD20 MAb or its fragment retains substantially the **B-cell** and **B-cell** lymphoma and leukemia cell targeting of the murine anti-CD20 MAb, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for:

(1) a humanized antibody or its fragment comprising the hA20Vk and hA2VH1 or hA2VH2;

(2) a chimeric anti-CD20 (cCD20) monoclonal antibody, (MAb) or its fragment comprising the CDRs of at least one murine anti-CD20 MAb variable region and the FRs of at least one murine anti-CD 20 MAb variable region;

(3) a human anti-CD20 (huD20)MAb;

(4) an **antibody fusion protein** or its fragment comprising at least two MAbs or their fragments, selected from the anti-CD20 MAbs cited above;

(5) a DNA sequence comprising a nucleic acid encoding a MAb or its fragment selected from the anti-CD20 MAb or their fragments and the **antibody fusion protein** cited above;

(6) an expression vector comprising the DNA sequence;

(7) a host cell comprising the DNA sequence or the expression vector;

(8) a **method** for expressing an anti-CD20 MAB, **antibody fusion protein** or their fragment;

(9) a B-lymphoma and leukemia cell targeting diagnostic or therapeutic conjugate comprising an antibody component comprising the anti-CD20 MAb, **antibody fusion protein** or their fragment that **binds** to the cell, where the antibody component is bound to at least one diagnostic or at least one therapeutic agent;

(10) a **method** for treating or diagnosing a **B-cell** lymphoma or leukemia or an **autoimmune disease** in a subject; and

(11) a **method** for pretargeting a cell in a patient suffering from a **B-cell** lymphoma or leukemia or an **autoimmune disease.**

ACTIVITY - Cytostatic; **Immunomodulator**; Dermatological; Antiinflammatory; Antiarthritic. No biological data given.

MECHANISM OF ACTION - Gene therapy.

USE - The **antibodies, fusion proteins** and conjugates are useful for diagnosing or preventing **B-cell** lymphoma, leukemia or an **autoimmune disease** (claimed), e.g. thrombocytopenia, lupus or rheumatoid arthritis.

Dwg.0/12

L23 ANSWER 2 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN

ACCESSION NUMBER: 2003-671467 [63] WPIDS

DOC. NO. CPI: C2003-183179

TITLE: **Method** of inhibiting RNA function involves contacting RNA molecule or cells having RNA with a specific compound and its salts, yohimbine, usnic acid,

or an acetamide.

DERWENT CLASS: B02
INVENTOR(S): DU, Z; FUJINAGA, K; GUY, R K; JAMES, T L; LIND, K E; MADRID, P; MAYER, M; PETERLIN, M B
PATENT ASSIGNEE(S): (REGC) UNIV CALIFORNIA
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003062388	A2	20030731	(200363)*	EN	54

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003062388	A2	WO 2003-US1688	20030116

PRIORITY APPLN. INFO: US 2002-349769P 20020116
AN 2003-671467 [63] WPIDS
AB WO2003062388 A UPAB: 20031001
NOVELTY - **Method** of inhibiting RNA function involves contacting an RNA molecule or cells comprising RNA with a compound (I) or its salts, yohimbine, usnic acid or N-(4-(2,5-dioxo-1-(4-trifluoromethoxy-phenyl)-pyrrolidin- 3-yl)-phenyl)-2,2,2-trifluoro-acetamide.

DETAILED DESCRIPTION - **Method** of inhibiting RNA function involves contacting an RNA molecule or cell comprising RNA with a compound of formula (I) or its salts, yohimbine, usnic acid, or N-(4-(2,5-dioxo-1-(4-trifluoromethoxy-phenyl)-pyrrolidin-3-yl)-phenyl)-2,2,2-trifluoro-acetamide.

A = O, NR1, S(O)n, CR2R3 or -A'-CR4R5;

A' = O, NR1, S(O)n or CR6R7;

n = 0-2;

B = NR, O, S or CR8R9; and

R1-R9, R = H or optionally substituted aliphatic or heteroaliphatic groups; and

ring D, ring E = optionally substituted 5-7 membered rings containing C, N, S and O atoms, where each ring includes at least one double bond; provided that:

(i) when B = NR, then NR and 2 C atoms on the ring adjacent to the N atom together comprise a ring of formula -NC=CC(OH)=C(COCH3)-C(O)-; and

(ii) at least one of A and B comprises an O, S or N ring atom.

INDEPENDENT CLAIMS are also included for:

(1) a **method** of increasing or decreasing the production of a protein by contacting a target mRNA molecule that encodes the protein with (I);

(2) a compound of formula (II);

(3) a pharmaceutical composition comprising (I), (II), or a compound of formula (III), that inhibits viral or microbial RNA function, or viral or microbial infection; and

(4) a complex formed between (I), (II) or (III) and the transactivation response element (TAR) site of HIV RNA.

R30 = (CH2)3R12;
R10 = halo or 1-4C alkoxy;
R11 = H or Me;
R12 = -N(CH3)2, N(CH2CH2)OH, -N(n-C4H9), -N(CH2C6H5), piperidine or 2-imidazole;
R20 = H, or aliphatic, heteroaliphatic or cycloheteroaliphatic ring (all optionally substituted), a ring of formula -NC=CC(OH)=C(COCH3)-C(O) (all carbon atoms on the side rings of the compound are optionally substituted by one or more OR', =O, =S, =NR', =NOR', NR'R'', SR', halo, SiR'R''R''', OC(O)R', C(O)R', CO2R', CONR'R'', OC(O)NR'R'', NR''C(O)R', NR'C(O)NR''R''', NR''C(O)2R', NRC(NRR'R'')=NR''', R'C(NR'R'')=NR''', NRC(NR'R'')=NR''', S(O)R', S(O)2R', S(O)2NR'R'', NRSO2R', CN or NO2); and
R'-R''' = H, halo, acyl, optionally substituted heteroaliphatic groups, unsubstituted aryl, aryl (substituted by 1-3 halo), optionally substituted aliphatic, optionally substituted oxyaliphatic groups, optionally substituted thioaliphatic groups, or aryl-1-4C aliphatic groups;
provided that:
(1) R11 = H if R10 = halo, and H or Me if R10 = alkoxy; and
(2) R12 = -N(CH3)2 if R10 is alkoxy, or -N(CH3)2, N(CH2CH2)OH, -N(n-C4H9), -N(CH2C6H5), piperidine or 2-imidazole, if R10 is halo.

ACTIVITY - Hemostatic; Nootropic; Neuroprotective; Antiarteriosclerotic; Cytostatic; Antiinflammatory; **Immunosuppressive**; Antidiabetic; Anorectic; Antiparkinsonian; Anti-HIV; Hepatotropic; Antipyretic; Protozoacide; Antidiarrheic; Antibacterial; Antitussive; Virucide; Fungicide; Antiparasitic.

MECHANISM OF ACTION - Inhibitor of RNA function.

The inhibitory effect of prochlorperazine (Ia) on RNA function was tested in HeLa cells. HeLa cells were preincubated with (Ia) (0.01-10 micro M). Two different sets of targets and effectors were used. HIV-1 long terminal repeat (LTR) and Tat were co-expressed. The heterologous tethering system of the regulator of expression of virion genes (Rev) and its Rev response element (RRE) RNA were utilized as a control. A test compound blocks Tat transactivation through TAR, and do not have any effects on the heterologous tethering of the RevTat fusion protein through RRE. DNA constructs containing the engineered chloramphenicol acyl transferase (**CAT**) gene preceded by TAR or RRE promoters were transfected into the HeLa cells with Lipofectin. Cells were incubated at 37 deg. C, 5 % CO2 for 5 hours. Cells were rinsed, fresh **drug** and media (3 ml 10% fetal calf serum (FCS), Dulbecco's modified Eagle medium (DMEM)) added, and then incubated for three days at 37 deg. C, 5 % CO2. Cells were collected by rinsing with 1 micro l phosphate buffer and centrifuged for 4 minutes at 14000 rpm. The supernatant was heated to 65 deg. C for 5 minutes and centrifuged 10 minutes at 14000 rpm. 100 micro l of supernatant, 1 mg chloramphenicol, 1 micro g 3H-acetyl-CoA, and EconoFluor solution were mixed, and immediately placed into the scintillation counter. **CAT** enzyme activity was measured by detecting the amount of 3H-acetyl-chloramphenicol. Prochlorperazine (Ia) (5-20 micro M) inhibited Tat transactivation up to 6-fold in a dose-dependent fashion. This compound had no effect on the hybrid RevTat protein on the RRE. Significant cellular toxicity resulted with greater than 20 micro M (Ia).

USE - The **method** is useful for inhibiting RNA function, preferably viral RNA function e.g. retroviral RNA (HIV), polio RNA, rhinoviral RNA, enteroviral RNA, or hepatitis C RNA; or microbial RNA such as bacterial RNA function. Also useful for inhibiting fungal or protozoal RNA function, for inhibiting microbial infection (bacterial infection), or viral infection e.g., retroviral infection (AIDS), a polio viral infection, rhinoviral infection enteroviral infection or hepatitis C infection. The compound inhibits HIV RNA function by **binding** to

the TAR site of the RNA. **Method** (1) is useful for increasing or decreasing the production of a protein that interferes with the progression of a **disease** associated with decreasing or increasing the production of the protein, respectively. **Method** (1) is preferably useful for increasing or decreasing the production of a protein that interferes with the progression of a **disease** such as amyloidosis, hemophilia, Alzheimer's **disease**, atherosclerosis, cancer, gigantism, dwarfism, hypothyroidism, hyperthyroidism, inflammation, cystic fibrosis, **autoimmune disorders**, diabetes, aging, obesity, neurodegenerative **disorders** or Parkinson's **disease**; and **diseases** caused by a bacteria, fungus, protozoa or virus such as HIV infection, AIDS, human **T-cell** leukemia, SIV infection, FIV infection, feline leukemia, hepatitis A, hepatitis B, hepatitis C, Dengue fever, malaria, rotavirus infection, severe acute gastroenteritis, diarrhea, encephalitis, hemorrhagic fever, syphilis, legionella, whooping cough, gonorrhea, sepsis, influenza, pneumonia, Tinea infection, Candida infection or meningitis (claimed).

DESCRIPTION OF DRAWING(S) - The figure shows a 3-dimensional structure of transactivation response element (TAR)-acetylpromazine complex, showing the relationship of an acetylpromazine molecule to TAR RNA.

Dwg.4/4

L23 ANSWER 3 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-468354 [44] WPIDS
DOC. NO. CPI: C2003-124924
TITLE: New alkane diol derivatives, useful in the treatment of, e.g. osteoporosis, rheumatoid arthritis, Pagets **disease**, inflammatory bowel syndrome, psoriasis, pulmonary alveolitis, hay fever or ulcerative colitis.
DERWENT CLASS: B05
INVENTOR(S): ARMOUR, K J; GREIG, I R; RALSTON, S H; VAN'T HOF, R J
PATENT ASSIGNEE(S): (UYAB-N) UNIV ABERDEEN
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003037321	A1	20030508	(200344)*	EN	195

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003037321	A1	WO 2002-GB4933	20021031

PRIORITY APPLN. INFO: GB 2001-26157 20011031
AN 2003-468354 [44] WPIDS
AB WO2003037321 A UPAB: 20030710
NOVELTY - Alkane diol derivatives (I), their salt, solvate, amide, ester, ether, chemically protected form or **prodrugs** are new.

DETAILED DESCRIPTION - Alkane diol derivatives of formula (I) their salt, solvate, amide, ester, ether, chemically protected form or **prodrug** are new.

A = 2-10C alkylene;

R1 = hydroxy protecting group; and

R2 = H or hydroxy protecting group.

The hydroxy groups of alkane diol are primary or secondary.

An INDEPENDENT CLAIM is also included for a compound of formula (IV) or its salt, solvate, amide, ester, ether, chemically protected form or **prodrug**.

J = H, halo, ONO2, ether, phosphonic acid or Ca2+ **binding** group; and

A, R1 = Definitions as above.

ACTIVITY - Osteopathic; Antiarthritic; Cytostatic; Antiinflammatory; Immunostimulant; Antirheumatic; Dermatological; Antiallergic; Hepatotropic; Antithyroid; Vasotropic; Antiulcer; Virucide; Antianemic; **Immunosuppressive**; Cardiant; Antipruritic.

MECHANISM OF ACTION - Osteoclast inhibitors; Bone resorption inhibitors.

The compounds were tested for their ability to inhibit osteoclast formation activity in the murine co-culture system.

1,4-butanediol mono (biphenyl-4-carboxylic acid) ester gave IC50 of 3.5 micromolar.

USE - (I) are used for the manufacture of a medicament useful in the treatment of conditions mediated by osteoclasts and/or bone resorption (particularly osteoporosis, rheumatoid arthritis, cancer associated bone **disease**, Paget's **disease** and/or conditions associated with inflammation or activation of the immune system of human or animal body by therapy) (claimed).

Also useful in the treatment of:

(1) multiple myeloma, hematological cancer, allergic **diseases**, such as atopy, allergic rhinitis, atopic dermatitis, anaphylaxis, allergic bronchopulmonary aspergillosis, and hypersensitivity pneumonitis (pigeon breeder's **disease**, farmer's lung **disease**, humidifier lung **disease**, malt worker's lung **disease**);

(2) allergies, including flea allergy dermatitis in mammals such as **domestic animals**, e.g. **dogs** and cats, contact allergens including mosquito bites or other insect sting allergies, poison ivy, poison oak, poison sumac, or other skin allergens;

(3) **autoimmune disorders**, type I diabetes, Crohn's **disease**, multiple sclerosis, arthritis, systemic lupus erythematosus, autoimmune (Hasimoto's) thyroiditis, autoimmune liver **diseases** such as hepatitis and primary biliary cirrhosis, hyperthyroidism (Graves' **disease**; thyrotoxicosis), insulin-resistant diabetes, autoimmune adrenal insufficiency (Addison's **disease**), autoimmune oophoritis, autoimmune orchitis, autoimmune hemolytic anemia, paroxysmal cold hemoglobinuria, Behcet's **disease**, autoimmune thrombocytopenia, autoimmune neutropenia, pernicious anemia, pure red cell anemia, autoimmune coagulopathies, myasthenia gravis, experimental allergic encephalomyelitis, autoimmune polyneuritis, pemphigus and other bullous **diseases**, rheumatic carditis, Goodpasture's syndrome, postcardiotomy syndrome, Sjogren's syndrome, polymyositis, dermatomyositis, or scleroderma; and

(4) **disease** states resulting from inappropriate inflammation, either local or systemic, for example, irritable or inflammatory bowel syndrome, skin **diseases** such as psoriasis and lichen planus, delayed type hypersensitivity, chronic pulmonary inflammation, e.g. pulmonary alveolitis and pulmonary granuloma, gingival inflammation or other periodontal **disease**, and osseous inflammation associated with lesions of endodontic origin,

hypersensitivity lung **diseases** such as hypersensitivity pneumonitis, and inflammation related to histamine release from basophils such as hay fever, histamine release from **mast cells** or **mast cell** tumors, types of type 1 hypersensitivity reactions (anaphylaxis, skin allergy, hives, allergic rhinitis, and allergic gastroenteritis) or ulcerative colitis.

ADVANTAGE - The compounds can be used both in vitro and in vivo with improved activity, efficacy, specificity, reduced toxicity, fewer undesired side-effects, simpler **methods** of administration, reduced dosage amount and reduced frequency of administration.

The compounds can also be synthesized, purified, handled or stored easily and economically.

(I) have excellent solubility properties especially in vivo, offering greater therapeutic benefit.

Dwg.0/19

L23 ANSWER 4 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-290078 [28] WPIDS
DOC. NO. NON-CPI: N2003-230679
DOC. NO. CPI: C2003-075401
TITLE: Identifying agents active against **autoimmune diseases** e.g. systemic lupus erythematosus, by determining the ability of a test agent to modulate the activity of the OBF-1 protein or gene, or Aiolos protein or gene.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): MATTHIAS, P D; SUN, J
PATENT ASSIGNEE(S): (NOVS) NOVARTIS FORSCHUNGSSTIFTUNG ZWEIGNIEDERL
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003018836	A2	20030306	(200328)*	EN	49

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003018836	A2	WO 2002-EP9365	20020821

PRIORITY APPLN. INFO: GB 2001-20441 20010822
AN 2003-290078 [28] WPIDS
AB WO2003018836 A UPAB: 20030501
NOVELTY - Identifying (M1) an agent active against an **autoimmune disease**, e.g. systemic lupus erythematosus (SLE), involves determining the ability of a test agent (I) to modulate e.g. to downregulate, the activity of OBF-1 protein (a B-lymphocyte specific activator of octamer site mediated gene transcription) or gene, or determining the ability of (I) to modulate the activity of Aiolos protein or gene.

DETAILED DESCRIPTION - Identifying (M1) an agent active against an **autoimmune disease**, involves:

(a) providing cells or extract from the cells containing OBF-1 protein, its fragment, variant or derivative, and a nucleic acid comprising a nucleotide sequence encoding a reporter gene (II) functionally linked to an OBF-1 responsive nucleotide sequence (RNS), contacting the cells or the extract with (I) in vitro, and determining the level of expression of the (II) by comparison to a control where the cells are not contacted with (I);

(b) providing OBF-1 protein, its fragment, variant or derivative, oct-1 protein and oct-2 protein, and a nucleic acid comprising a nucleotide sequence encoding (II) functionally linked to an OBF-1 RNS, and subjecting the OBF-1, oct-1, oct-2 and the nucleic acid construct together to in vitro transcription in the presence or absence of (I), and determining the level of expression of (II) in the presence or absence of (I);

(c) providing cells containing a nucleic acid comprising a nucleotide sequence encoding (II) functionally linked to an Aiolos RNS, contacting the cells with (I) in vitro, and determining the level of expression of (II) by comparison to a control where the cells are not contacted with (I), where the cells do not contain an Aiolos protein;

(d) providing OBF-1 protein, its fragment, variant or derivative, providing an oct protein selected from oct-1, oct-2 or POU domain of the oct-1 or oct-2 protein, combining the OBF-1 protein with the oct protein in the presence or absence of (I), and determining **binding** of the OBF-1 protein to the oct protein in the presence or absence of (I);

(e) providing cells containing OBF-1 protein, its fragment, variant or derivative, an above said oct protein, preparing an extract from the cells, mixing the extract with a labeled nucleic acid probe containing an oct-1 or oct-2 protein **binding** site, e.g. an octomer site, in the presence or absence of (I) and determining the formation of a complex between the OBF-1 protein, the oct protein and the nucleic acid probe in the presence or absence of (I);

(f) providing cells containing an OBF-1 gene, contacting the cells in vitro with (I), and determining the expression of OBF-1 gene as compared to a level of expression in the absence of (I);

(g) providing cells containing Aiolos gene, contacting the cells in vitro with (I), and determining the expression of Aiolos gene as compared to the level of expression in the absence of (I);

(h) administering (I) to an Aiolos deficient mouse and determining at least one effect of (I) on symptoms of SLE in the mouse;

(i) administering (I) to an Aiolos deficient mouse, extracting **B cells** or **B cell** precursors from the mouse, culturing the **B cells** in vitro and determining at least one effect of (I) on the **B cells**; or

(j) contacting Aiolos deficient **B cells** with (I) in vitro and determining at least one effect of (I) on **B cells**.

INDEPENDENT CLAIMS are also included for:

(1) screening (M2) an agent capable of modulating the activity of Aiolos protein, by providing cells containing an Aiolos protein, a nucleic acid comprising a nucleotide sequence encoding (II) functionally linked to an Aiolos RNS, contacting the cells with (I) in vitro, and determining the level of expression of (II) by comparison to a control where the cells are not contacted with (I);

(2) diagnosing a pre-disposition to **autoimmune disease**, e.g. SLE, by determining in vitro all or part of the amino acid sequence of Aiolos protein or gene in a sample from an individual;

(3) an Aiolos protein (III), its derivative or fragment for use as a pharmaceutical;

(4) a nucleic acid (IV) encoding (III) for use in therapy;
(5) a pharmaceutical composition (PC) for preventing or treating an **autoimmune disease**, e.g. SLE, comprising (III), its fragment, variant or derivative, or (IV);
(6) use of an Aiolos deficient mouse for the identification of an agent active against an **autoimmune disease** for e.g. SLE; and
(7) use of Aiolos deficient **B cells** in vitro for the identification of an agent active against an **autoimmune disease**, for e.g. SLE.
ACTIVITY - **Immunosuppressive**; Dermatological; Antiinflammatory.
MECHANISM OF ACTION - Modulator of activity of OBF-1 or Aiolos protein or gene.
No biological data given.
USE - M1 is useful for identifying an agent active against an **autoimmune disease** e.g. SLE. (III) is useful in as a pharmaceutical. (IV) is useful in therapy. (III) and (IV) are useful in the manufacture of a medicament for treating an **autoimmune disease**, e.g. SLE and in the treatment of **autoimmune disease**. PC is useful for the prevention and treatment of **autoimmune disease** e.g. SLE (claimed).
Dwg.0/5

L23 ANSWER 5 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-615866 [58] WPIDS
DOC. NO. CPI: C2003-167963
TITLE: New medium for expanding natural killer cells expressing CD56+CD3- phenotype comprising CellGro (Trademark) SCGM, for treating e.g. allergic or **autoimmune diseases**, immunodeficiency, or solid tumors.
DERWENT CLASS: B04 D16
INVENTOR(S): DILBER, M S
PATENT ASSIGNEE(S): (DILB-I) DILBER M S
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2003068306	A1	20030410	(200358)*		14

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2003068306	A1 Provisional	US 2001-318871P	20010914
		US 2002-242788	20020913

PRIORITY APPLN. INFO: US 2001-318871P 20010914; US 2002-242788 20020913
AN 2003-615866 [58] WPIDS
AB US2003068306 A UPAB: 20030910
NOVELTY - A new medium for expanding natural killer cells expressing the CD56+CD3- phenotype comprising CellGro SCGM to which interleukin (IL) 2 and anti-CD3 antibodies has been added, is new.
DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a **method** for expanding natural killer cells expressing the CD56+CD3- phenotype, where the natural killer cells are isolated from a monolayer cell concentrate, washed, and suspended and incubated in a medium

comprising CellGro SCGM defined above.
ACTIVITY - Cytostatic; Immunostimulant; Antiallergic; **Immunosuppressive**; Antibacterial; Antianaemic.
No suitable biological data given.
MECHANISM OF ACTION - Cell therapy.
No suitable biological data given.
USE - The expanded natural killer cells can be used for curative or prophylactic treatment of patients with recurrent malignant **disease** following allogeneic stem cell transplantation, patients undergoing autologous stem cell transplantation for cancer, patients with severe infections after allogeneic or autologous stem cell transplantation, or patients with hematological malignancies, recurrent or acute infections, allergic or **autoimmune diseases**, immunodeficiency, or solid tumors (claimed).
Dwg.0/7

L23 ANSWER 6 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-571360 [54] WPIDS
DOC. NO. CPI: C2003-154442
TITLE: Generating genetically modified vertebrate precursor lymphocytes for producing any heterologous antibody or binding protein comprises effecting differentiation of the precursor lymphocytes into mature lymphoid lineage cells.
DERWENT CLASS: B04 D16
INVENTOR(S): GRAWUNDER, U; MELCHERS, G F
PATENT ASSIGNEE(S): (GRAW-I) GRAWUNDER U; (MELC-I) MELCHERS G F
COUNTRY COUNT: 101
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1321477	A1	20030625	(200354)*	EN	111
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
WO 2003068819	A1	20030821	(200356)#	EN	
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZW					

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1321477	A1	EP 2001-130805	20011222
WO 2003068819	A1	WO 2001-EP15303	20011222

PRIORITY APPLN. INFO: EP 2001-130805 20011222; WO 2001-EP15303 20011222
AN 2003-571360 [54] WPIDS
AB EP 1321477 A UPAB: 20030821
NOVELTY - Generating vertebrate lymphocytes that can be used for the production of any heterologous antibody, antigen receptor, artificial binding protein, or their functional fragments, comprising:
(a) genetically modifying vertebrate precursor lymphocytes; and
(b) effecting differentiation of the precursor lymphocytes into

mature lymphoid lineage cells either in vitro or in vivo, is new.

DETAILED DESCRIPTION - Generating vertebrate lymphocytes that can be used for the production of any heterologous antibody, antigen receptor, artificial binding protein, or their functional fragments, comprising:

(a) genetically modifying vertebrate precursor lymphocytes, which:

(a) are derived from primary lymphoid organs; and

(b) have the potential to differentiate into mature lymphoid lineage cells by introducing at least one exogenous genetic element encoding at least one heterologous antibody, antigen receptor, artificial binding protein, or their fragments; and

(b) effecting differentiation of the precursor lymphocytes into mature lymphoid lineage cells either in vitro or in vivo, thus, generating lymphocytes capable of producing the heterologous antibody, antigen receptor, artificial binding protein, or their fragments, is new.

INDEPENDENT CLAIMS are included for the following:

(1) genetically modified vertebrate precursor lymphocytes and more mature lymphoid lineage cells derived from the precursor lymphocytes, obtained by the **method**;

(2) immortalized cells producing heterologous antibodies, antigen receptor, artificial binding protein, or their fragments;

(3) vector or genetic constructs for carrying out the **method** ; and

(4) a pharmaceutical or diagnostic preparation, comprising at least one antibody, antigen receptor, artificial binding protein, or their fragments, obtained by the **method**, displaying either wild-type immune effector functions, or modified or artificial effector functions not derivable from germline encoded heterologous immunoglobulins or antigen receptors.

USE - The **method** and the genetically modified and differentiated vertebrate lymphocytes are useful in the production of any heterologous antibody, artificial binding protein, antigen receptor, or their fragments, where the antibody is monoclonal or polyclonal, or partially resembles a human antibody, binding protein or antigen receptor (claimed). The antibodies are useful for the diagnosis, prevention and treatment of **diseases**.

ADVANTAGE - The **method** combines the advantages of both the phage display system (i.e. speed and flexibility in generating human antibodies, and the ability to modify and improve the properties of existing antibodies), and of the human immunoglobulin transgenic mouse technology (i.e. the ability to obtain high affinity antibodies due to affinity maturation occurring in the immune system, and the production antibodies with physiologic and natural structural features).

Dwg.0/15

L23 ANSWER 7 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN

ACCESSION NUMBER: 2003-058723 [05] WPIDS

DOC. NO. CPI: C2003-015207

TITLE: New ion channel array for evaluating ion channel expression profiles of a biological material, comprises ion channel polynucleotide spots stably associated with a solid support.

DERWENT CLASS: B04 D16

INVENTOR(S): JENSEN, B S; JENSEN, J B; KJAER, K; MADSEN, L S

PATENT ASSIGNEE(S): (NEUR-N) NEUROSEARCH AS

COUNTRY COUNT: 100

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002086050	A2	20021031	(200305)*	EN	53

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002086050 A2		WO 2002-DK253	20020418

PRIORITY APPLN. INFO: DK 2001-635 20010420
AN 2003-058723 [05] WPIDS
AB WO 200286050 A UPAB: 20030121
NOVELTY - An ion channel array comprising a multiplicity of individual ion channel polynucleotide spots stably associated with a surface of a solid support, is new. The individual ion channel polynucleotide spot comprises an ion channel polynucleotide composition having a non-conserved region of an ion channel polynucleotide family member, the spots representing at least two different regions of an ion channel polynucleotide family member.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) preparing the novel array, comprising:

(a) generating the non-conserved regions of ion channel polynucleotide family members;

(b) preparing a multiplicity of compositions each comprising at least a non-conserved region; and

(c) stably associating the compositions in individual spots on a surface of a solid support;

(2) a set of primers specific for the non-conserved regions cited above, which are used for the production of the array;

(3) determining an ion channel polynucleotide expression profile in a biological material, comprising:

(a) obtaining a polynucleotide sample from the biological material;

(b) labeling the sample to obtain a labeled target polynucleotide sample;

(c) contacting at least one labeled target polynucleotide sample with the array under conditions sufficient to produce a hybridization pattern; and

(d) detecting the hybridization pattern to obtain the ion channel polynucleotide expression profile of the biological material;

(4) a diagnostic **method** for determining a difference in ion channel polynucleotide expression profiles from at least a first and a second different biological material, comprising:

(a) obtaining a first ion channel expression profile of the first biological material;

(b) obtaining a second ion channel expression profile of the second biological material; and

(c) comparing the first and the second expression profiles to identify any difference between the two ion channel expression profiles;

(5) identifying a therapeutic, prophylactic and/or toxic agent involved in a direct or indirect action on the ion channel expression profile in a biological material, comprising:

(a) employing steps (a) and (b) of **method** (4);

(b) applying a test compound to the second biological material and

obtaining a third ion channel expression profile;

(c) comparing the first, second and third ion channel expression profiles; and

(d) identifying any differences in the expression profiles to identify any biological response of the test compound on the ion channel expression profile; and

(6) an ion channel kit for use in a hybridization assay, comprising the novel ion channel array.

USE - The array is useful in evaluating ion channel expression profiles of a biological material. The **methods** are used in producing the array and in identifying a therapeutic, prophylactic and/or toxic agent involved in a direct or indirect action on the ion channel expression profile. The kit is used in hybridization assays.

Dwg.0/0

L23 ANSWER 8 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2003-075446 [07] WPIDS
DOC. NO. CPI: C2003-019527
TITLE: Kit, useful for targeting of diagnostic or therapeutic agent to target site in mammal, comprises first conjugate, optionally clearing agent and second conjugate.
DERWENT CLASS: B04 B05 D16 K08
INVENTOR(S): HNATOWICH, D J; LIU, G; RUSCKOWSKI, M
PATENT ASSIGNEE(S): (UYMA-N) UNIV MASSACHUSETTS
COUNTRY COUNT: 100
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002078638	A2	20021010	(200307)*	EN	43
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG US UZ VN YU ZA ZM ZW					
US 2003003102	A1	20030102	(200309)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002078638	A2	WO 2002-US9749	20020401
US 2003003102	A1 Provisional	US 2001-279809P	20010330
	Provisional	US 2001-341794P	20011221
		US 2002-112094	20020401

PRIORITY APPLN. INFO: US 2001-341794P 20011221; US 2001-279809P 20010330; US 2002-112094 20020401
AN 2003-075446 [07] WPIDS
AB WO 200278638 A UPAB: 20030129
NOVELTY - A kit comprising:

(a) a first conjugate;

(b) optionally a clearing agent; and

(c) a second conjugate;

is new.

(a) comprises a targeting moiety and a Morpholino oligomer (d). (c)

comprises a complementary (d), and a diagnostic or therapeutic agent. The targeting moiety **binds** selectively to a primary, target-specific **binding** site of the target site or to a substance produced by or associated with the target site.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a **method** of delivering a diagnostic or therapeutic agent to a target site in a mammal, comprising:

(1) administering (a);

(2) optionally administering (b) to clear non-localized (a) from circulation; and

(3) then administering (c).

ACTIVITY - Cytostatic; Antiinflammatory; **Immunosuppressive.**

USE - For targeting a diagnostic or therapeutic agent to a target site in a mammal, useful for internal detection or treatment of tumors or other lesions, infectious diseases, inflammatory diseases, and **autoimmune diseases** (claimed), and for tumor localization/imaging.

ADVANTAGE - The kit is more specific, affordable, inexpensive, and provides higher uptake and lower uptake in normal tissues. The kit can be prepared from inexpensive starting materials, but provides better specificity, stability, predictable targeting and/or more antigen-antibody effects.

Dwg.0/8

L23 ANSWER 9 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN

ACCESSION NUMBER: 2002-489999 [52] WPIDS

DOC. NO. CPI: C2002-139110

TITLE: New **immunomodulatory** peptides from heat shock proteins, useful for treating immunological **disorder** in subjects such as humans, e.g. **autoimmune disease** (e.g. arthritis), infectious **disease**, inflammatory bowel **disease** or cancer.

DERWENT CLASS: B04 D16

INVENTOR(S): ALBANI, S; CARSON, D A; MARTINI, A; PRAKKEN, B J

PATENT ASSIGNEE(S): (MART-I) MARTINI A; (REGC) UNIV CALIFORNIA; (ALBA-I) ALBANI S; (CARS-I) CARSON D A; (PRAK-I) PRAKKEN B J

COUNTRY COUNT: 98

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002036611	A2	20020510	(200252)*	EN	84
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2002020038	A	20020515	(200258)		
US 2003031679	A1	20030213	(200314)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002036611	A2	WO 2001-US45344	20011031
AU 2002020038	A	AU 2002-20038	20011031
US 2003031679	A1 Provisional	US 2000-245181P	20001101
		US 2001-1938	20011031

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002020038 A	Based on	WO 2002036611

PRIORITY APPLN. INFO: US 2000-245181P 20001101; US 2001-1938 20011031

AN 2002-489999 [52] WPIDS

AB WO 200236611 A UPAB: 20020815

NOVELTY - A peptide, which is an immunogenic portion derived from a dnaJ heat shock protein (hsp), comprising any of 26 amino acid sequences fully defined in the specification, is new.

DETAILED DESCRIPTION - A peptide comprising any of sequences P1-P26, which is an immunogenic portion derived from a dnaJ hsp, is new.

(1) P1: Gln-Asp-Tyr-Tyr-Glu-Ile-Leu-Gly-Val-Ser-Lys-Thr-Ala-Glu-Glu;

(2) P2: Arg-Lys-Ala-Tyr-Lys-Arg-Leu-Ala-Met-Lys-Tyr-His-Pro-Asp-Arg;

(3) P3: Gln-Lys-Arg-Ala-Ala-Tyr-Asp-Gln-Tyr-Gly-His-Ala-Ala-Phe-Glu-Gln;

(4) P4: Gln-Gly-Phe-Phe-Ala-Val-Gln-Gln-Thr-Cys-Pro-His-Cys-Gln-Gly;

(5) P5: Ser-Lys-Thr-Leu-Ser-Val-Lys-Ile-Pro-Gly-Ala-Val-Asp-Thr-Gly;

(6) P6: Gly-Asp-Leu-Tyr-Val-Gln-Val-Gln-Val-Lys-Gln-His-Pro-Ile-Phe;

(7) P7: Tyr-Cys-Glu-Val-Pro-Ile-Asn-Phe-Ala-Met-Ala-Ala-Leu-Gly-Gly;

(8) P8: Pro-Ile-Asn-Phe-Ala-Met-Ala-Ala-Leu-Gly-Gly-Glu-Ile-Glu-Val;

(9) P9: Asn-Ser-Tyr-Tyr-Glu-Ile-Leu-Asp-Val-Pro-Arg-Ser-Ala-Ser-Ala;

(10) P10: Lys-Asp-Tyr-Tyr-Gln-Thr-Leu-Gly-Leu-Ala-Arg-Gly-Ala-Ser-Asp;

(11) P11: Thr-Thr-Tyr-Tyr-Asp-Val-Leu-Gly-Val-Lys-Pro-Asn-Ala-Thr-Gln;

(12) P12: Lys-Lys-Ala-Tyr-Arg-Arg-Lys-Ala-Leu-Gln-Trp-His-Pro-Asp-Lys;

(13) P13: Lys-Arg-Ala-Tyr-Arg-Arg-Gln-Ala-Leu-Arg-Tyr-His-Pro-Asp-Lys;

(14) P14: Lys-Lys-Ala-Tyr-Arg-Lys-Leu-Ala-Leu-Lys-Tyr-His-Pro-Asp-Lys;

(15) P15: Phe-Arg-Ser-Val-Ser-Thr-Ser-Thr-Thr-Phe-Val-Gln-Gly-Arg-Arg;

(16) P16: Pro-Gly-Met-Val-Gln-Gln-Ile-Gln-Ser-Val-Cys-Met-Glu-Cys-Gln;

(17) P17: Gly-Arg-Arg-Ile-Thr-Thr-Arg-Arg-Ile-Met-Glu-Asn-Gly-Gln-Glu;

(18) P18: Gln-Ala-Tyr-Glu-Val-Leu-Ser-Asp-Ala-Lys-Lys-Arg-Glu-Leu-Tyr-Asp;

(19) P19: Glu-Ala-Tyr-Glu-Val-Leu-Ser-Asp-Lys-His-Lys-Arg-Glu-Ile-Tyr-Asp;

(20) P20: Ser-Gly-Pro-Phe-Phe-Thr-Phe-Ser-Ser-Ser-Phe-Pro-Gly-His-Ser;

(21) P21: Asp-Gly-Gln-Leu-Lys-Ser-Val-Thr-Ile-Asn-Gly-Val-Pro-Asp-Asp;

(22) P22: Asp-Leu-Gln-Leu-Ala-Met-Ala-Tyr-Ser-Leu-Ser-Glu-Met-Glu-Ala;

(23) P23: Glu-Asp-Leu-Phe-Met-Cys-Met-Asp-Ile-Gln-Leu-Val-Glu-Ala-Leu;

(24) P24: Leu-Cys-Gly-Phe-Gln-Lys-Pro-Ile-Ser-Thr-Leu-Asp-Asn-Arg-Thr;

(25) P25: Arg-Thr-Ile-Val-Ile-Thr-Ser-His-Pro-Gly-Gln-Ile-Val-Lys-His; and

(26) P26: Gly-Arg-Leu-Ile-Ile-Glu-Phe-Lys-Val-Asn-Phe-Pro-Glu-Asn-Gly.

INDEPENDENT CLAIMS are also included for the following:
(1) modulating an immune response in a subject by administering the immunogenic peptide portion of a dnaJ hsp to the subject;
(2) modulating immunoeffector cell responsiveness by contacting immunoeffector cells of a subject with the peptide portion of a dnaJ hsp cited above;
(3) a chimeric polypeptide comprising the peptide operatively linked to at least one heterologous polypeptide;
(4) a polynucleotide encoding the peptide;
(5) a recombinant nucleic acid molecule comprising the polynucleotide above operatively linked to at least one heterologous nucleotide sequence;
(6) a vector comprising the polynucleotide; and
(7) a cell containing the polynucleotide.
ACTIVITY - Immunomodulator; Cytostatic; Antiinflammatory; Antibacterial; Antiarthritic. No relevant biological data given.
MECHANISM OF ACTION - Interferon-Stimulator-Gamma; Tumor Necrosis Factor-Stimulator-Alpha; Interleukin-Stimulator-1; Interleukin-Stimulator-6; Interleukin-Stimulator-12; Interleukin-Stimulator-23; Interleukin-Inhibitor-4, Interleukin-Inhibitor-10, Transforming Growth Factor-Inhibitor-Beta; Interferon-Inhibitor-Gamma; Tumor Necrosis Factor-Inhibitor-Alpha; Interleukin- Inhibitor-1; Interleukin- Inhibitor-6; Interleukin- Inhibitor-12; Interleukin- Inhibitor-23; Interleukin-Stimulator-4, Interleukin- Stimulator-10, Transforming Growth Factor-Stimulator-Beta; Vaccine.
USE - The immunogenic peptide is useful for modulating (i.e. augmenting/inducing or reducing/inhibiting) an immune response in a subject having an immunological disorder (e.g. autoimmune disease), an infectious disease, an inflammatory bowel disease or cancer. The autoimmune disease is arthritis, specifically an articular juvenile idiopathic arthritis. The immunogenic peptide is also useful for modulating immunoeffector cell responsiveness in a subject (claimed). The immunogenic peptide is particularly useful for treating the above-mentioned diseases in mammals, e.g. cat, dog, horse, farm animal (e.g. ovine, bovine or porcine) or human. In general, the peptide is useful in methods involving mucosal tolerization, DNA vaccination, anergy induction or active immunization.
Dwg.0/26

L23 ANSWER 10 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-454647 [48] WPIDS
DOC. NO. CPI: C2002-129311
TITLE: Novel control region of delta-5-desaturase gene useful as a target for screening compounds useful in the treatment of **diseases** involving abnormal lipid metabolism including diabetic neuropathy.
DERWENT CLASS: B04 D16
INVENTOR(S): ALLEN, S J; DE ANTUENO, R J; HAARDT, M; JENKINS, D K; KNICKLE, L C; NWAKA, S O; PONTON, A; WINTHER, M D
PATENT ASSIGNEE(S): (XENO-N) XENON GENETICS INC
COUNTRY COUNT: 98
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002034940	A2	20020502	(200248)*	EN	93

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PH PL PT RO

RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2002014856 A 20020506 (200257)
EP 1354047 A2 20031022 (200370) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002034940	A2	WO 2001-CA1520	20011026
AU 2002014856	A	AU 2002-14856	20011026
EP 1354047	A2	EP 2001-983344	20011026
		WO 2001-CA1520	20011026

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2002014856	A	Based on	WO 2002034940
EP 1354047	A2	Based on	WO 2002034940

PRIORITY APPLN. INFO: US 2000-243009P 20001026
AN 2002-454647 [48] WPIDS
AB WO 200234940 A UPAB: 20020730
NOVELTY - An isolated human delta -5-desaturase (hD5D) control region (I) comprising a DNA sequence (S1) of 1357 bp defined in the specification, fragment of (S1) having 15 nucleotides, a sequence which is 90% homologous with (S1), or its hybridizable sequence, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated rat delta -5-desaturase (rD5D) coding portion of the fatty acid desaturase gene, comprising a DNA sequence (S2) of 1344 bp defined in the specification, fragment of (S2) having 15 nucleotides, a sequence which is 90% homologous with (S2), or its hybridizable sequence;

(2) an isolated polypeptide (II) comprising a sequence of 447 amino acids defined in the specification, its fragment of 15 amino acids, or its 90% homolog or its salt;

(3) a recombinant nucleic acid molecule (III) comprising a control region of a mammalian D5D and a reporter gene, where the control region is transcriptionally linked to the reporter gene so as to effectively initiate, terminate or regulate transcription of the reporter gene;

(4) a vector construct (IV) comprising (III), capable of expressing the reporter gene to generate a reporter gene product that can be detected upon introduction of the vector construct into an appropriate host cell or host system;

(5) a host cell (V) or system transformed or transfected with (IV);

(6) a modulator (VI) identified by a screening **method** utilizing (V);

(7) a pharmaceutical composition (VII) comprising (VI);

(8) a rat D5D protein (VIII) having a linked tag sequence;

(9) an antibody immunoreactive with (II), a polypeptide of a human D5D, or their immunogenic portion, where the antigen is produced in a host system;

(10) screening for a modulator which is capable of modulating the enzymatic activities of functional mammalian D5D and/or delta -6-desaturase (D6D) enzymes within the same host system, by providing a host system containing nucleic acid sequences, which encode both mammalian D5D and D6D enzymes operably associated with promoter region effective to initiate, terminate or regulate a level of expression of the nucleic acid

sequence, contacting the host system with a test component, simultaneously evaluating the enzymatic activities of the D5D and D6D, where a measurable difference in a level of lipid metabolites or associated cofactors in the presence of the test component compared to a control under identical conditions but in the absence of the test component is an indicator of the ability of the test component to modulate D5D and/or D6D enzyme activity; and selecting as the modulator a test component which exhibits ability;

(11) screening (M) for a modulator which is capable of modulating the enzymatic activities of functional mammalian D5D, by utilizing a host system containing a nucleic acid sequence encoding a mammalian D5D operably associated with a promoter region; and

(12) the use of n-propyl gallate for the treatment of a **disease** (D) such as arterial hypertension, hypercholesterolemia, atherosclerotic heart **disease**, chronic inflammatory **disorders**, **autoimmune disorders**, allergic eczema and other atopic **disorders**, inflammatory process such as rheumatoid arthritis, diminished lymphocyte proliferation, **T-cell**-mediated cytotoxicity, natural killer cell activity, macrophage-mediated cytotoxicity, monocyte and neutrophil chemotaxis, major histocompatibility class II expression and antigen presentation, production of pro-inflammatory **cytokines** (interleukins 1 and 6, tumor necrosis factor) and adhesion molecule expression, eczema, psoriasis, acute respiratory distress syndrome (ARDS), articular cartilage degradation (ACD) and cancer.

ACTIVITY - Antidiabetic; Hypotensive; Antilipemic; Antiatherosclerotic; Cardiant; Antiinflammatory; **Immunosuppressive**; Antiallergic; Dermatological; Antirheumatic; Antiarthritic; Antipsoriatic; Cytostatic.

No supporting data is given.

MECHANISM OF ACTION - Modulator of lipid metabolism; modulator of transcriptional expression or enzymatic activity of mammalian D5D enzyme.

USE - (VI) is useful for treating lipid metabolism abnormalities, human diabetic neuropathy, and **diseases** indicated as (D) above. (V) is useful for screening for a modulator capable of modulating or regulating the transcriptional expression of a mammalian D5D gene, especially for identifying modulators that modulate lipid metabolism or diabetic neuropathy. The screening **method** is an assay for identifying modulators that modulate the n-3 lipid metabolic pathway, conversion of 18:3n3-22:6n3, or n-9 lipid metabolic pathway, conversion of 16:0-22:4n9 or n-6 lipid metabolic pathway, conversion of 18:2n6-22:5n6 (claimed). (I) provides a powerful tool for dissecting the role of D5D gene expression and inducing modifications, which eliminate or control alterations associated with metabolic **disorders**.

Dwg.0/15

L23 ANSWER 11 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-148002 [19] WPIDS
DOC. NO. CPI: C2002-045991
TITLE: Composition useful for treating rheumatic **disease** and immune system **disorders** e.g. diabetes mellitus, graft-related **disease**, good pasture's syndrome, comprises soluble cytotoxic T lymphocyte A4 mutant molecule.
DERWENT CLASS: B04 B05 D16
INVENTOR(S): BECKER, J; CARR, S; COHEN, R; HAGERTY, D; PEACH, R J
PATENT ASSIGNEE(S): (BRIM) BRISTOL-MYERS SQUIBB CO; (BECK-I) BECKER J; (CARR-I) CARR S; (COHE-I) COHEN R; (HAGE-I) HAGERTY D; (PEAC-I) PEACH R J
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002002638	A2	20020110	(200219)*	EN	128
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001073174	A	20020114	(200237)		
NO 2002006264	A	20030219	(200321)		
US 2003083246	A1	20030501	(200331)		
KR 2003017606	A	20030303	(200345)		
HU 2003001727	A2	20030929	(200369)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002002638	A2	WO 2001-US21204	20010702
AU 2001073174	A	AU 2001-73174	20010702
NO 2002006264	A	WO 2001-US21204	20010702
		NO 2002-6264	20021227
US 2003083246	A1 Provisional	US 2000-215913P	20000703
		US 2001-898195	20010702
KR 2003017606	A	KR 2003-700018	20030102
HU 2003001727	A2	WO 2001-US21204	20010702
		HU 2003-1727	20010702

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001073174	A Based on	WO 2002002638
HU 2003001727	A2 Based on	WO 2002002638

PRIORITY APPLN. INFO: US 2000-215913P 20000703; US 2001-898195 20010702

AN 2002-148002 [19] WPIDS

AB WO 200202638 A UPAB: 20020321

NOVELTY - A pharmaceutical composition (I) comprising a soluble cytotoxic T lymphocyte antigen 4 (CTLA4) mutant molecule (II) and a carrier for treating rheumatic **disease**, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a kit comprising soluble (II) for treating rheumatoid arthritis.

ACTIVITY - Antirheumatic; Antiarthritic; Analgesic; Dermatological; Antiinflammatory; Antidiabetic; **Immunosuppressive**; Neuroprotective; Antiulcer; Antipsoriatic; Cytostatic; Nephrotropic; Thyromimetic; Antianemic.

L104EA29YIg was tested for antirheumatic and antiarthritic activity. A total of 214 patients, including 54 males and 160 females were randomized into groups of 25 to 32 patients per treatment group. 32 patients received a placebo, 92 received L104EA29YIg, and 90 received CTLA4Ig. The patients who followed protocol guidelines and did not discontinue before day 57 received a total of 4 intravenous infusions, one infusion each on days 1, 15, 29 and 57. All patients were evaluated on days 1, 15, 29, 43, 57, 71 and 85. The doses administered included 0.5, 2.0, or 10.0 mg/kg of L104EA29YIg (denoted as LEA.5, LEA2 and LEA10) or of CTLA4Ig (denoted as CTLA.5, CTLA2 and CTLA10). Patients were evaluated for

baseline symptoms of **disease** activity prior to and after receiving any infusions. These baseline evaluations included joint swelling, joint tenderness, inflammation, morning stiffness, **disease** activity, especially soluble interleukin (IL)-2r and C-reactive protein levels. Results showed that the percent of patients having reduced swollen and tender joint counts compared to the patients having no response to treatment with CTLA4Ig, L104EA29YIg, or placebo, and the therapeutic response appeared to be dose-dependent. After treatment, soluble IL-2r levels were -2 %, -10 %, and -22 % for CTLA4IG and -4 %, -18 %, and -32 % for L104EA29YIg at 0.5, 20.0 and 10.0 mg/kg respectively, compared to +3 % for the placebo. C-reactive protein levels were +12 %, -15 % and -32 % for CTLA4Ig and +47 %, -33 % and -47 % for L104EA29YIg at 0.5, 2.0 and 10.0 mg/kg respectively, compared to +20 % for the placebo.

MECHANISM OF ACTION - Inhibits the **binding** of B7 molecule to CTLA4 and/or CD28 on **T cells; T-cell**/B7-positive cell interactions blocker (claimed).

USE - (I) is useful for treating rheumatic **disease** especially rheumatoid arthritis; and for inducing a pathophysiological change associated with rheumatic **disease** which is reduced structural damage in a subject which is a human, monkey, ape, **dog**, **cat**, cow, **horse**, rabbit, mouse, or rat, where (I) specifically **binds** to a B7 molecule. The **method** further administering an **immunosuppressive** agent such as corticosteroids, nonsteroidal antiinflammatory **drugs**, cyclosporin prednisone, azathioprine, methotrexate, tumor necrosis factor (TNF)- alpha blockers or antagonists, inflixamib, hydroxychloroquine, sulphasalazine, gold salts, etanercept, or anakinra, and for alleviating a symptom associated with a rheumatic **disease** from joint swelling, pain, tenderness, morning stiffness, structural damage; an elevated level of serum C-reactive protein, soluble interleukin (IL)-2r, soluble ICAM-1, soluble E-selection and erythrocyte sedimentation rate. (All claimed). (I) optionally with other pharmaceutical agents is useful for treating immune system **disorder** which include **autoimmune diseases** e.g. systemic lupus erythematosus, Addison's **disease**, diabetes mellitus, multiple sclerosis, Crohn's **disease**, ulcerative colitis, Sjogren's syndrome, scleroderma, sympathetic ophthalmia; graft-related **disease** e.g. graft-versus-host **disease**; immunoproliferative **diseases** e.g. psoriasis, **T cell** lymphoma, Hashimoto's thyroiditis, pernicious anemia, good pasture's syndrome.

Dwg.0/33

L23 ANSWER 12 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-351262 [38] WPIDS
CROSS REFERENCE: 1999-045130 [04]; 2001-122704 [01]
DOC. NO. CPI: C2002-099668
TITLE: Treating **B-cell, T-cell, myeloid cell, mast cell** or **plasma cell disorders** such as malignancy, involves administering an antibody.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): GOLDENBERG, D M
PATENT ASSIGNEE(S): (GOLD-I) GOLDENBERG D M
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2002041847	A1	20020411	(200238)*		17

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2002041847 A1	CIP of	US 1998-38995	19980312
	CIP of	US 1999-307816	19990510
		US 2001-921290	20010803

FILING DETAILS:

PATENT NO	KIND	PATENT NO
US 2002041847 A1	CIP of	US 6134982
	CIP of	US 6306393

PRIORITY APPLN. INFO: US 2001-921290 20010803; US 1998-38995 19980312; US 1999-307816 19990510

AN 2002-351262 [38] WPIDS

CR 1999-045130 [04]; 2001-122704 [01]

AB US2002041847 A UPAB: 20020709

NOVELTY - Treating a **B-cell, T-cell, myeloid cell, mast cell,** or **plasma cell disorder** in a **domestic animal**, comprising administering an antibody specific for an antigen or epitope on one of the cells, is new.

ACTIVITY - **Immunosuppressive**; Cytostatic.

A 65 pound, 7 year old male golden retriever diagnosed with diffuse large cell aggressive lymphomase was placed on a combination chemotherapy with vincristine, cyclophosphamide, prednisolone, and doxorubicin, with good response. The **dog** was subsequently characterized with progressive lymphadenopathy, and seven months later found to have extensive lymphoma infiltration of bone marrow, extensive lymphoadenopathy of neck, chest abdomen and pelvis, and hepatosplenomegaly. The **dog** was given therapy with 120 mg 1F5 monoclonal antibody, intravenously, by infusion. The treatment was repeated weekly for 4 weeks. Four months after the final dose, a computerized tomography scan of the **dog** shows no signs of lymphoma.

MECHANISM OF ACTION - None given.

USE - For treating **B-cell, T-cell, myeloid cell, mast cell,** or **plasma cell disorders**, particularly a malignancy, or **autoimmune disorder**, in a **domestic animal**, such as a **dog, cat** or **horse** (claimed).

Dwg.0/0

L23 ANSWER 13 OF 29 MEDLINE on STN

ACCESSION NUMBER: 2002472841 MEDLINE

DOCUMENT NUMBER: 22219878 PubMed ID: 12235516

TITLE: Rapid hematopoietic engraftment following fractionated TBI conditioning and transplantation with CD34(+) enriched hematopoietic progenitor cells from partially mismatched related donors.

AUTHOR: Redei I; Langston A A; Lonial S; Cherry J K; Allen A J; Hamilton E; Jones M; Bartlett V M; Waller E K

CORPORATE SOURCE: Emory University School of Medicine, Winship Cancer Institute, Department of Hematology and Oncology, Bone Marrow and Stem Cell Transplant Center, Emory University, Atlanta, GA, USA.

SOURCE: BONE MARROW TRANSPLANTATION, (2002 Sep) 30 (6) 335-40.
Journal code: 8702459. ISSN: 0268-3369.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200307
ENTRY DATE: Entered STN: 20020918
Last Updated on STN: 20030703
Entered Medline: 20030702

AB Nineteen adult patients with poor-risk hematologic **malignancy** received **T cell**-depleted (TCD) hematopoietic progenitor cell (HPC) transplant from partially mismatched related donors (PMRD). The preparative regimen (FITFA) included fractionated TBI, thiotepa, fludarabine, and **horse** (n = 3) or rabbit (n = 16) anti-thymocyte anti-sera (ATG). GVHD prophylaxis consisted of TCD by positive/negative selection using the Isolex 300i system and pre-transplant ATG with no post-transplant **immunosuppression.** The mean number (+/-s.d.) of transplanted CD34(+) and CD3(+) cells were 8.9 x 10(6)/kg +/-4.3 (range 2.6-19.3) and 1.4 x 10(4)/kg +/-1.2 (range 0.3-4.6) respectively. Seventeen patients evaluable for neutrophil engraftment achieved an ANC >0.5 x 10(9)/l at a median of 12 days (range 9-27), with evidence of full donor chimerism. Thirteen patients died of the following causes: relapse (n = 6), infections (n = 5), interstitial pneumonia (n = 1), and unknown causes (n = 1) None of the recipients of rabbit ATG required therapy for acute or chronic GVHD. Five patients are alive and **disease**-free at a median time of 303 days post transplant (range 100-660). The FITFA preparative regimen using fractionated TBI is well tolerated and is sufficiently **immunosuppressive** to allow rapid and stable donor origin hematopoietic engraftment without 'mega' doses of CD34(+) cells. Combination of stringent ex vivo TCD and pre-transplant ATG is effective GVHD prophylaxis.

L23 ANSWER 14 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:465240 BIOSIS
DOCUMENT NUMBER: PREV200200465240
TITLE: **Immunosuppressive** and antiinflammatory effects of triptolide and its **prodrug** PG-490-88.
AUTHOR(S): Chen, Benny J.; Chao, Nelson J. [Reprint author]
CORPORATE SOURCE: Bone Marrow Transplantation Program, Duke University Medical Center, 2400 Pratt St., Suite 1100, Durham, NC, 27705, USA
SOURCE: Drugs of the Future, (January, 2002) Vol. 27, No. 1, pp. 57-60. print.
ISSN: 0377-8282.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Sep 2002
Last Updated on STN: 4 Sep 2002

L23 ANSWER 15 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:143303 BIOSIS
DOCUMENT NUMBER: PREV200300143303
TITLE: Epitope Spreading in Induced and Spontaneous Equine Recurrent Uveitis.
AUTHOR(S): Deeg, C. A. [Reprint Author]; Thurau, S. R.; Gerhards, H.; Kaspers, B. [Reprint Author]; Wildner, G.

CORPORATE SOURCE: Institute of Animal Physiology, LMU, Munich, Germany
SOURCE: ARVO Annual Meeting Abstract Search and Program Planner, (2002) Vol. 2002, pp. Abstract No. 1532. cd-rom.
Meeting Info.: Annual Meeting of the Association For Research in Vision and Ophthalmology. Fort Lauderdale, Florida, USA. May 05-10, 2002.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Mar 2003
Last Updated on STN: 19 Mar 2003

AB Purpose: Immunization with a multideterminant native antigen leads to induction of the **T cell** response to one or more of its dominant determinants. There is no reaction to other regions of the same antigen, therefore these determinants are cryptic epitopes. **T cells** specific for these cryptic endogenous epitopes activated during acute inflammation could play the major effector role in subsequent relapses of autoimmune uveitis, as it was demonstrated for other **autoimmune diseases** as EAE or autoimmune diabetes. The goal of our study was the investigation of the **T cell** response of **horses** with IRBP induced uveitis (HEU) to various S-Antigen and IRBP derived peptides at several timepoints during the uveitic attack and subsequent induced relapses. **Methods**: We induced uveitis in **horses** by two s.c. injections of IRBP emulsified in CFA. Relapses were induced by two subsequent immunizations at day 56 and 84. Control **horses** received CFA only. In vitro proliferation of PBL was analyzed at day 0, 2, 4, 6, 8 and 12 after each immunization of experimental and control **horses**. Results: All **horses** developed uveitis at day 6-7 after the second immunization. Intramolecular epitope spreading to other IRBP epitopes was recognized after the second immunization in all **horses** of the experimental group. Novel reactivity was observed after every subsequent immunization, combined with a loss of reactivity to some of the peptides that were recognized at the beginning. More interestingly, we also found intermolecular epitope spreading to S-Ag in 5 out of 7 **horses** after the second immunization. At that timepoint peripheral blood derived lymphocytes proliferated in response to S-Ag derived peptides such as PD-SAg, peptide M and SAg 281. We could also observe intramolecular and intermolecular epitope spreading in **horses** with spontaneous equine recurrent uveitis. Conclusion: These findings underscore the possibility that the progression of autoimmune uveitis could be perpetuated by a shifting of **T cell** autoreactivity from a single predominant determinant to other uveitogenic epitopes.

L23 ANSWER 16 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2003:357130 BIOSIS
DOCUMENT NUMBER: PREV200300357130
TITLE: Soluble CD22, a New Test Which Correlates with **Disease** Status in **B-Cell** Leukemia and Lymphoma.
AUTHOR(S): Kreitman, Robert J. [Reprint Author]; Wilson, Wyndham H. [Reprint Author]; Margulies, Inger [Reprint Author]; Pastan, Ira [Reprint Author]; Matsushita, Kakushi [Reprint Author]
CORPORATE SOURCE: Laboratory of Molecular Biology, National Cancer Institute, Bethesda, MD, USA
SOURCE: Blood, (November 16, 2002) Vol. 100, No. 11, pp. Abstract No. 2996. print.
Meeting Info.: 44th Annual Meeting of the American Society of Hematology. Philadelphia, PA, USA. December 06-10, 2002.

American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Aug 2003
Last Updated on STN: 18 Sep 2003

AB The combined incidence of non-Hodgkin's lymphoma (NHL) and acute and chronic lymphocytic leukemias (ALL and CLL) is 65,000 new cases/year in the US, with a death rate of 30,000/year. The prevalence of NHL alone in the US was estimated at apprx300,000 ten years ago. The availability of effective therapy for several decades has resulted in a large number of patients with varying levels of remission and **disease** burden. Knowledge of overall **disease** burden is important in planning effective therapy and in diagnosing relapse early. Several blood tests have been developed to measure **disease** burden in hematologic malignancies, including those detecting soluble CD25 (sCD25) or CD30, but these are of limited use in most **B-cell malignancies** due to lack of overexpression of these antigens. To determine whether CD22, an antigen found on the surface of most **B-cell malignancies**, could be detected in a soluble form in the serum, an enzyme-linked immunosorbent (ELISA) assay was developed utilizing 2 anti-CD22 monoclonal antibodies (MAbs). One MAb was used to coat plates and **bind** sCD22 from serum, and the other MAb was biotinylated so that it would **bind** both sCD22 and avidin conjugated to **horseradish** peroxidase. A fusion containing sCD22 and human Fc (immunoglobulin CH2 + CH3 domains) was constructed, expressed in human 293 **T cells**, and purified from serum-free medium using sizing chromatography to >80% purity by western blot. Using the standard curve, sCD22 was measurable from 0.019 to 10 ng/ml, and recovery of exogenous sCD22 added to normal serum was 85-115%. Precision was high with inter-and intraplate variations within 10%. In normal donors, the sCD22 concentrations were 0.5-1.22 ng/ml, with a mean +/- standard deviation (SD) of 0.90 +/- 0.28 ng/ml (n=9). The pretreatment levels of sCD22 were 1.72-69.6 ng/ml (28.76 +/-18.21 ng/ml, n=23) in hairy cell leukemia (HCL)($p < 0.0001$) and 2.41-14.5 ng/ml (19.6 +/- 14.5 ng/ml, n=16) in chronic lymphocytic leukemia (CLL)($p < 0.0001$). After achievement of complete remission (CR) with the recombinant **immunotoxin** BL22, the sCD22 levels in HCL patients fell to 0.84 +/- 0.5 ng/ml (n=11), compared to 11.1 +/- 18.4 ng/ml in 5 patients not achieving CR. In 2 responding CLL patients, sCD22 fell from 22.8 to 2.1 ng/ml and from 4.7 to 0.7 ng/ml. sCD22 correlated with **disease** burden in terms of spleen size or circulating malignant count ($p < 0.0001$), and was more useful than sCD25 assessment, particularly in patients with CD25-negative **diseases**. sCD22 levels in 12 patients with **B-cell** HIV-related NHL were 1.0-6.9 ng/ml before and < 0.5 ng/ml after responding to multiagent chemotherapy ($p < 0.0001$), and later increased in 3 out of 3 patients diagnosed with **disease** progression. We conclude that sCD22 is a potentially valuable test of **disease** burden in patients with **B-cell malignancies** and additional studies are underway to further define its clinical utility.

L23 ANSWER 17 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-114392 [15] WPIDS
DOC. NO. CPI: C2002-035137
TITLE: Soluble **T cell** receptor fusion or conjugate complexes useful in treating malignant **disorders** comprises either **T**

cell receptor and polypeptide connected by a peptide linker or molecules covalently bound to a carrier.

DERWENT CLASS: B04 D16
INVENTOR(S): CARD, K F; WEIDANZ, J A; WONG, H C
PATENT ASSIGNEE(S): (SUNO-N) SUNOL MOLECULAR CORP
COUNTRY COUNT: 96
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001093913	A2	20011213	(200215)*	EN	66

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2001075246	A	20011217	(200225)		
EP 1289564	A2	20030312	(200320)	EN	

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

US 2003144474	A1	20030731	(200354)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001093913	A2		WO 2001-US18145	20010605
AU 2001075246	A		AU 2001-75246	20010605
EP 1289564	A2		EP 2001-941935	20010605
			WO 2001-US18145	20010605
US 2003144474	A1	Provisional	US 2000-209536P	20000605
			US 2001-874907	20010605

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001075246	A	Based on	WO 2001093913
EP 1289564	A2	Based on	WO 2001093913

PRIORITY APPLN. INFO: US 2000-209536P 20000605; US 2001-874907 20010605
AN 2002-114392 [15] WPIDS
AB WO 200193913 A UPAB: 20020306
NOVELTY - Soluble **T cell** receptor fusion or conjugate complex, comprising either **T cell** receptor (TCR) (I) and a polypeptide connected by a peptide linker (II) or several molecules covalently bound to a carrier (III), is new. (I) and (II) have different recognition **binding** sites. (III) has at least one remaining free amine group. The carrier is covalently bound to a single chain **T cell** receptor.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) preparing soluble **T cell** receptor fusion or conjugate complex, comprising:

(a) providing **T cell** receptor chain or its sub-fragment;

(b) providing the polypeptide corresponding to a second chain or its

sub-fragment;
(c) connecting the **T cell** receptor chain and the second chain to a peptide linker; and
(d) recovering the linked **T cell** receptor fusion polypeptide complex, thus generating a **T cell** receptor fusion complex;
(2) preparing soluble **T cell** receptor fusion or conjugate complex, comprising:
(a) reacting a polymer carrier which has covalently bound several molecules with a **T cell** receptor chain; and
(b) reductively stabilizing the resulting conjugate molecule;
(3) a nucleic acid sequence encoding the **T cell** receptor fusion complex comprising (I) and (II).
ACTIVITY - Cytostatic; Antiinflammatory; **Immunosuppressive**; Antiviral; Neuroprotective; Antidiabetic; Antirheumatic; Antiarthritic.
MECHANISM OF ACTION - TCR **binder**.
CTLL-2 cells were hydrodiethidium labeled and incubated for 20 minutes at room temperature (RT) with an equal number of calcein-AM labeled T2 cells pulsed with either 50 micro g of 149 or 246 peptide. The conjugation was observed between cells when 1 micro g of fusion protein was added to the incubation mixture containing CTLL-2 cells and 264 peptide-loaded T2 cells (i.e. 3.25 %) while conjugate formulation was not observed with mixture comprising the 149 peptide pulsed T2 cells used (i.e. 0.88 %).
USE - In therapeutic composition for treating **disorders** e.g. malignant **disorder**, **autoimmune disorder**, inflammatory response, viral infection; as **diagnostic composition**; for imaging studies (claimed). The complex is also used for the treatment of allergies and **autoimmune diseases** e.g. multiple sclerosis, insulin-dependent diabetes mellitus and rheumatoid arthritis, and in targeting particular tumor antigens in human patients. it can be used for the treatment of cancer e.g. hepatitis C virus (HCV), human immunodeficiency virus (HIV), etc, for veterinary applications e.g. treatment of **disorders** of livestock e.g. cattle, sheep, etc and pets such as **dog** and cats, and to guide, target or direct localized toxic agents to specific sites to intervene in a **disease** process.
ADVANTAGE - The **T cell** complexes teaches the use of genetic fusions and chemical conjugation as **methods** for effecting such linkage. The (TCR)-based reagents provides higher killing efficiency of tumor cells, recognizes many potential tumor antigens as exposed on the surface of the cells or accessible to the molecule. The antigens recognized by antibodies are not heterogeneic in nature, thus does not limit the effectiveness of the antibody to a single tumor histology.
Dwg.0/13

L23 ANSWER 18 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2002-122025 [16] WPIDS
DOC. NO. NON-CPI: N2002-091558
DOC. NO. CPI: C2002-037342
TITLE: Identifying modulators of **T cell** receptor (TCR) and major histocompatibility complex (MHC) antigen immune complexes, useful for treating immune **disorders**, by determining if a test compound alters specific TCR-MHC **binding**.
DERWENT CLASS: B04 D16 S03
INVENTOR(S): ACEVEDO, J; BURKHARDT, M; CARD, K F; RHODE, P; TAL, R; WEIDANZ, J A; WITTMAN, V; WONG, H C
PATENT ASSIGNEE(S): (SUNO-N) SUNOL MOLECULAR CORP

COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001090747	A2	20011129	(200216)*	EN	207
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2001061624	A	20011203	(200221)		
EP 1287363	A2	20030305	(200319)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
CN 1430728	A	20030716	(200363)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001090747	A2	WO 2001-US15699	20010516
AU 2001061624	A	AU 2001-61624	20010516
EP 1287363	A2	EP 2001-935537	20010516
		WO 2001-US15699	20010516
CN 1430728	A	CN 2001-810020	20010516

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001061624	A	Based on	WO 2001090747
EP 1287363	A2	Based on	WO 2001090747

PRIORITY APPLN. INFO: US 2000-206920P 20000525
AN 2002-122025 [16] WPIDS
AB WO 200190747 A UPAB: 20020308
NOVELTY - Identifying compounds that modulate an immune complex, of a **T cell** receptor (TCR) and a major histocompatibility complex (MHC) antigen, comprising determining if a test compound alters specific **binding** between the TCR and the MHC antigen molecules, is new.

DETAILED DESCRIPTION - Identifying compounds that modulate an immune complex comprising a TCR and a MHC antigen comprising:

(a) contacting a first TCR molecule and MHC antigen molecule in the presence or absence of at least one test compound, the contacting being under conditions sufficient to **bind** the TCR and the MHC antigen molecules specifically as an immune complex;

(b) detecting presence of the immune complex in the presence and absence of the test compound;

(c) selecting a test compound that alters specific **binding** between the TCR and MHC antigen molecules; and

(d) identifying the selected compound as being capable of modulating the immune complex.

The **method** also comprises:

(a) contacting cells expressing a first TCR molecule with an MHC antigen molecule bound to a solid support in the presence or absence of a test compound, the contacting being under conditions sufficient to **bind** the TCR and the bound MHC antigen molecule specifically as an

immune complex;
(b) detecting presence of the immune complex in the presence and absence of the test compound by measuring at least one response from the cells;
(c) selecting a test compound that alters specific **binding** between the first TCR and bound MHC antigen molecules; and
(d) identifying the selected compound as being capable of modulating the immune complex.
INDEPENDENT CLAIMS are also included for the following:
(1) a test compound identified by the novel **method**;
(2) a pharmaceutical composition comprising at least one of the compound of (1);
(3) a **method** of inhibiting or stimulating an immune response in a mammal comprising administering a therapeutic amount of the compound of (1)
(4) a kit for performing the novel **method**; and
(5) a recombinant TCR molecule comprising a mammalian CD3 zeta sequence or its functional fragment.
ACTIVITY - **Immunomodulator**; **immunosuppressive**; antiallergic.
No biological data is given.
MECHANISM OF ACTION - None given.
USE - For providing high throughput screening assays for detecting and identifying compositions or compounds which are useful for modulating an immune response, especially in a mammal. The compounds are particularly useful for treating immune **disorders**, e.g. **autoimmune diseases** or allergies.
Dwg.0/24

L23 ANSWER 19 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-476199 [51] WPIDS
CROSS REFERENCE: 2001-442253 [47]; 2001-442255 [47]; 2001-451890 [48]; 2001-451908 [48]; 2001-451909 [48]; 2001-451912 [48]; 2001-451938 [48]; 2001-451939 [48]; 2001-457603 [49]; 2001-457740 [49]; 2001-465363 [50]; 2001-465571 [50]; 2001-465578 [50]; 2001-465705 [50]; 2001-476114 [51]; 2001-476164 [51]; 2001-476197 [51]; 2001-476198 [51]; 2001-476282 [51]; 2001-476283 [51]; 2001-483140 [52]; 2001-483233 [52]; 2001-488707 [53]; 2001-488788 [53]; 2001-488875 [53]; 2001-488895 [53]; 2001-496929 [54]; 2001-496930 [54]; 2001-496931 [54]; 2001-496932 [54]; 2001-514838 [56]; 2001-522358 [57]; 2001-565565 [63]; 2001-582152 [65]; 2001-582153 [65]; 2001-589862 [66]; 2001-589934 [66]; 2001-607699 [69]; 2001-611724 [70]; 2001-611725 [70]; 2001-626375 [72]; 2001-626426 [72]; 2001-626432 [72]; 2001-626527 [72]; 2001-639362 [73]; 2002-010428 [01]; 2002-025688 [03]; 2002-062370 [08]; 2002-280918 [32]; 2002-575369 [61]; 2002-590824 [63]; 2002-674924 [72]; 2003-018710 [01]; 2003-028924 [02]; 2003-110596 [10]; 2003-174164 [17]; 2003-456302 [43]; 2003-678194 [64]; 2003-697229 [66]; 2003-697230 [66]; 2003-697231 [66]; 2003-810980 [76]
DOC. NO. CPI: C2001-142863
TITLE: Novel carcinoembryonic antigen-like protein, useful for treating breast, prostate and colon cancers, inflammatory and **autoimmune disorders**, as **immunosuppresant**, as decoy receptor in bacterial and viral infections.
DERWENT CLASS: B04 D16
INVENTOR(S): ARTERBURN, M C; BOYLE, B J; DRMANAC, R A; KUO, C; LIU, C;

TANG, Y T
PATENT ASSIGNEE(S): (HYSE-N) HYSEQ INC
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001055337	A2	20010802	(200151)*	EN	131

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001036553	A	20010807	(200174)	
EP 1276902	A2	20030122	(200308)	EN

R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001055337	A2	WO 2001-US2614	20010125
AU 2001036553	A	AU 2001-36553	20010125
EP 1276902	A2	EP 2001-908711	20010125
		WO 2001-US2614	20010125

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001036553	A	Based on	WO 2001055337
EP 1276902	A2	Based on	WO 2001055337

PRIORITY APPLN. INFO: US 2000-665533 20000919; US 2000-491404 20000125

AN 2001-476199 [51] WPIDS

CR 2001-442253 [47]; 2001-442255 [47]; 2001-451890 [48]; 2001-451908 [48]; 2001-451909 [48]; 2001-451912 [48]; 2001-451938 [48]; 2001-451939 [48]; 2001-457603 [49]; 2001-457740 [49]; 2001-465363 [50]; 2001-465571 [50]; 2001-465578 [50]; 2001-465705 [50]; 2001-476114 [51]; 2001-476164 [51]; 2001-476197 [51]; 2001-476198 [51]; 2001-476282 [51]; 2001-476283 [51]; 2001-483140 [52]; 2001-483233 [52]; 2001-488707 [53]; 2001-488788 [53]; 2001-488875 [53]; 2001-488895 [53]; 2001-496929 [54]; 2001-496930 [54]; 2001-496931 [54]; 2001-496932 [54]; 2001-514838 [56]; 2001-522358 [57]; 2001-565565 [63]; 2001-582152 [65]; 2001-582153 [65]; 2001-589862 [66]; 2001-589934 [66]; 2001-607699 [69]; 2001-611724 [70]; 2001-611725 [70]; 2001-626375 [72]; 2001-626426 [72]; 2001-626432 [72]; 2001-626527 [72]; 2001-639362 [73]; 2002-010428 [01]; 2002-025688 [03]; 2002-062370 [08]; 2002-280918 [32]; 2002-575369 [61]; 2002-590824 [63]; 2002-674924 [72]; 2003-018710 [01]; 2003-028924 [02]; 2003-110596 [10]; 2003-174164 [17]; 2003-456302 [43]; 2003-678194 [64]; 2003-697229 [66]; 2003-697230 [66]; 2003-697231 [66]; 2003-810980 [76]

AB WO 200155337 A UPAB: 20031125
NOVELTY - An isolated polypeptide (carcinoembryonic antigen (CEA)-like protein) (I) comprising an amino acid sequence which is at least 80% identical to a fully defined sequence of 425 (S4), 45, 45, 20, 405, 45 (S6-S10) amino acids as given in the specification, a mature protein or its extracellular portion or active domain, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a polypeptide (Ia) having CEA-like activity comprising 10 consecutive amino acids of (S4) and (S6)-(S10);

(2) an isolated polynucleotide (II) comprising a fully defined sequence of 416 (S2), 1557 (S3) or 1278 (S5) nucleotides as given in the specification, its translated protein coding portion, the mature protein coding portion, the extracellular portion, or active domain;

(3) an isolated polynucleotide encoding a polypeptide with biological activity, which hybridizes to the complement of (II) under stringent hybridization conditions;

(4) an isolated polynucleotide encoding a polypeptide with biological activity, where the polynucleotide has greater than 90% sequence identity with (II);

(5) an isolated polynucleotide which comprises the complement of (II);

(6) a vector comprising (II);

(7) an expression vector comprising (II);

(8) a host cell (III) genetically engineered to express (II);

(9) a composition comprising (I) and a carrier;

(10) a polynucleotide encoding (I) or (Ia);

(11) an antibody specific for (I);

(12) detecting (M1) (II) in a sample involves, contacting the sample with a compound that **binds** to and forms a complex with (II) to form a complex and detecting the complex, so that if a complex is detected, (II) is detected. The **method** alternately (M2) involves contacting the sample under stringent hybridization conditions with nucleic acid primers that anneal to (II), amplifying a product comprising at least a portion of (II) and detecting the product and thereby (II) in the sample;

(13) detecting (I) in a sample involves contacting the sample with a compound that **binds** to and forms a complex with (I) to form a complex and detecting the complex, so that if a complex is detected, (I) is detected;

(14) identifying a compound that **binds** to (I) involves contacting the compound with the polypeptide to form a polypeptide/compound complex and detecting the complex, so that if the polypeptide/compound complex is detected, a compound that **binds** to (I) is identified;

(15) the **method** alternately involves contacting the compound with (I), in a cell, to form a polypeptide/compound complex, where the complex drives expression of a reporter gene sequence in the cell and detecting the complex by detecting reporter gene sequence expression, so that if the polypeptide/compound complex is detected, a compound that **binds** to (I) is identified;

(16) preparation of (I);

(17) a kit comprising (I);

(18) a nucleic acid array (IV) comprising (II) or a unique segment of (II) attached to the surface;

(19) treating a subject in need of enhanced activity or expression of (I) involves administering an agonist of (I), (I) or a polynucleotide encoding (I) under conditions such that the polypeptide is produced, and a carrier; and

(20) treating a subject in need to inhibit activity or expression of (I) involves administering an antagonist of (I), a polypeptide that competes with (I) for its ligand or a polynucleotide that inhibits the expression of a nucleotide sequence encoding (I), and a carrier.

ACTIVITY - Cytostatic; antiinflammatory; **immunosuppressive**; antianemic; vulnerary; osteopathic; antiarthritic; antiulcer; nootropic; neuroprotective; cerebroprotective; immunostimulant; antirheumatoid;

antithyroid; virucide; contraceptive; antiinfertility; hemostatic; thrombolytic; anticoagulant; antibacterial; antiparkinsonian; vasotropic.

No supporting data is given.

MECHANISM OF ACTION - CEA-like protein expression or activity modulator; antisense therapy or gene therapy; cell development, proliferation, growth, differentiation, survival, regeneration, immune responses modulator.

USE - (II) is useful as hybridization probes, oligomers or primers, in computer readable media, chromosome and gene mapping for recombinant production of (I) and in generation of antisense DNA or RNA, their chemical analogs, etc. They are also useful as diagnostics. (II) can be used to induce immune responses. (I) is useful for generating antibodies, as molecular weight markers and as a food supplement. (I) can be used for in vitro biding assays to identify molecules which **bind** to the polypeptide.

(I) and (II) can be used for treating breast, prostate, colon and other cancers, **disorders** relating to inflammation and autoimmunity, as **immunosuppressant** in organ transplantations, as a decoy receptor in bacterial and viral infections. Detecting (I) or (II) is used as part of prognostic or diagnostic evaluation of **disorders** and for identifying subjects exhibiting predisposition to such conditions.

The novel polynucleotides can be used to express recombinant protein for analysis, characterization or therapeutic use, as markers for tissues in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage of tissue differentiation or development or in **disease** states), as molecular weight markers on Southern gels, as chromosome markers or tags (when labeled) to identify chromosomes or to map related gene positions, to compare with endogenous DNA sequences in patients to identify potential genetic **disorders** , as probes to hybridize and thus discover novel, related DNA sequences, as source of information to derive polymerase chain reaction (PCR) primers for genetic fingerprinting, as a probe to subtract-out known sequences in the process of discovering other novel polynucleotides, for selecting and making oligomers for attachment to a gene chip or other support, including for examination of expression patterns, to raise anti-DNA antibodies or elicit another immune response.

The novel proteins can similarly be used in assays to determine biological activity, including in a panel of multiple proteins for high throughput screening, to raise antibodies or to elicit another immune response, as a reagent (including the labeled reagent) in assays designed to quantitatively determine levels of the protein (or its receptor) in biological fluids, as markers for tissues in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage of tissue differentiation or development or in a **disease** state). Proteins involved in these **binding** interactions can also be used to screen for peptide or small molecular inhibitors or agonists of the **binding** reactions. The proteins can also be used for making antibody substances that are specifically immunoreactive with CEA-like proteins.

Dwg.0/2

L23 ANSWER 20 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-273585 [28] WPIDS
CROSS REFERENCE: 2001-367218 [38]
DOC. NO. CPI: C2001-083005
TITLE: Identifying a test compound that **binds** to a target RNA molecule, comprises contacting a dye-labeled target RNA molecule with test compound and determining structure of test compound of RNA test compound complex.

DERWENT CLASS: B04 D16
INVENTOR(S): HWANG, S; RANA, T M; TAMILARASU, N
PATENT ASSIGNEE(S): (UYNE-N) UNIV NEW JERSEY MEDICINE & DENTISTRY
COUNTRY COUNT: 95
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001025486	A1	20010412	(200128)*	EN	54
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW					
AU 2000078559	A	20010510	(200143)		
US 6420591	B1	20020716	(200248)		
EP 1218544	A1	20020703	(200251)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
US 6503713	B1	20030107	(200306)		
JP 2003511045	W	20030325	(200330)		67
US 6583309	B1	20030624	(200343)		
US 2003153523	A1	20030814	(200355)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2001025486	A1		WO 2000-US27389	20001004
AU 2000078559	A		AU 2000-78559	20001004
US 6420591	B1	Provisional	US 1999-157646P	19991004
			US 2000-679728	20001004
EP 1218544	A1		EP 2000-968684	20001004
			WO 2000-US27389	20001004
US 6503713	B1	Provisional	US 1999-157646P	19991004
			US 2000-679451	20001004
JP 2003511045	W		WO 2000-US27389	20001004
			JP 2001-528636	20001004
US 6583309	B1	Provisional	US 1999-157646P	19991004
		Div ex	US 2000-679728	20001004
			US 2002-151800	20020521
US 2003153523	A1	Provisional	US 1999-157646P	19991004
		Cont of	US 2000-679451	20001004
			US 2002-295761	20021115

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000078559	A	Based on	WO 2001025486
EP 1218544	A1	Based on	WO 2001025486
JP 2003511045	W	Based on	WO 2001025486
US 6583309	B1	Div ex	US 6420591
US 2003153523	A1	Cont of	US 6503713

PRIORITY APPLN. INFO: US 1999-157646P 19991004; US 2000-679728 20001004; US 2000-679451 20001004; US 2002-151800 20020521; US 2002-295761 20021115
AN 2001-273585 [28] WPIDS

CR 2001-367218 [38]
AB WO 200125486 A UPAB: 20030828
NOVELTY - Identifying a test compound (I) that **binds** to a target RNA molecule, comprising contacting a dye-labeled target RNA molecule with substantially one type of (I) attached to a solid support, thereby providing a dye-labeled target RNA:support-attached (I) complex and determining the structure of the substantially one type of (I) of the RNA:(I) complex, is new.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a compound (C) selected from the group consisting of:

(1) H2N-(L)Lys-(D)Lys-(L)Asn-OH;
(2) H2N-(L)Lys-(D)Lys-(D)Asn-OH;
(3) H2N-(L)Lys-(L)Lys-(L)Asn-OH;
(4) H2N-(L)Arg-(D)Lys-(L)Asn-OH;
(5) H2N-(L)Arg-(D)Lys-(L)Val-OH;
(6) H2N-(L)Arg-(D)Lys-(L)Arg-OH;
(7) H2N-(L)Thr-(D)Lys-(L)Asn-OH; and
(8) H2N-(D)Thr-(D)Lys-(L)Phe-OH.

ACTIVITY - Nootropic; neuroprotective; **immunosuppressive**; antiarteriosclerotic; hemostatic; cytostatic; antidiabetic; anorectic; antiparkinsonian; anti-human immunodeficiency virus (HIV); virucide; hepatotropic; antiinflammatory; protozoacide; antibacterial; antidiarrheic.

MECHANISM OF ACTION - Displaces ligand of target RNA (claimed); inhibits protein-RNA or RNA-RNA interaction.

Different amounts of tripeptide ID1 were added during transfection of pSV2-Tat and pAL plasmids (expressing first exon of Tat protein and luciferase enzyme, respectively) into HL3T1 cells. Increasing amounts of tripeptide ID1 resulted in a decrease of **CAT** (undefined) activity while luciferase activity was not affected. In the presence of 700 nM concentration of tripeptide ID1, more than 90% of Tat-transactivation was inhibited. The results showed that tripeptide ID1 **binds** trans-activation response region RNA (TAR RNA) and inhibits Tat-TAR interactions in vivo, and hence suitable for preventing or treating human immunodeficiency virus (HIV) infections or acquired immunodeficiency syndrome (AIDS) in patients.

USE - (I) is useful for forming a target RNA:test compound complex, for increasing or decreasing the production of a protein by contacting a target mRNA molecule that encodes the protein with (I), and for treating or preventing a **disease** e.g., amyloidosis, hemophilia, Alzheimer's **disease**, atherosclerosis, cancer, giantism, dwarfism, hypothyroidism, hyperthyroidism, inflammation, cystic fibrosis, **autoimmune disorders**, diabetes aging, obesity, neurodegenerative **disorders**, Parkinson's **disease**, human immunodeficiency virus (HIV) infection, acquired immunodeficiency syndrome (AIDS), human **T-cell** leukemia, simian immunodeficiency virus (SIV) infection, feline immunodeficiency virus (FIV) infection, feline leukemia, hepatitis B, hepatitis C, Dengue fever, malaria, rotavirus infection, severe acute gastroenteritis, diarrhea, encephalitis, hemorrhagic fever, syphilis, legionella, whooping cough, gonorrhea, sepsis, influenza, pneumonia, tinea infection, Candida infection and meningitis, whose progression is associated with in vivo **binding** of (I) to target RNA (claimed).

ADVANTAGE - The **method** is fast and efficient in screening combinatorial compound libraries for molecules that **bind** to RNAs and potentially disrupt protein-RNA and RNA-RNA interactions.
Dwg.0/5

L23 ANSWER 21 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-588994 [66] WPIDS

CROSS REFERENCE: 1999-395155 [33]; 2001-079409 [02]
DOC. NO. CPI: C2001-174601
TITLE: New triazine derivatives are angiogenesis inhibitors useful in the treatment of e.g. metastatic solid tumors, carcinomas of the breast, rectum, lung or colon, Crohn's **disease** and diabetic retinopathy.
DERWENT CLASS: B02 B03
INVENTOR(S): DAVIDSON, D J; HENKIN, J; MCCROSKEY, R W; SHEPPARD, G S; WOODS, K W
PATENT ASSIGNEE(S): (ABBO) ABBOTT LAB
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6288228	B1	20010911	(200166)*		20

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
US 6288228	B1	Provisional	US 1997-69592P	19971212
		Cont of	US 1998-209396	19981210
			US 2000-680383	20001005

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 6288228	B1	Cont of	US 6150362

PRIORITY APPLN. INFO: US 1997-69592P 19971212; US 1998-209396 19981210; US 2000-680383 20001005
AN 2001-588994 [66] WPIDS
CR 1999-395155 [33]; 2001-079409 [02]
AB US 6288228 B UPAB: 20011113
NOVELTY - 1,3,5-Triazine derivatives (I) and their salts and **prodrugs** are new.
DETAILED DESCRIPTION - 1,3,5-Triazine derivatives of formula (I) and their salts and **prodrugs** are new.
R1 - R4 = H, 1-20C alkyl or 1-20C alkanoyl; or
NR1R2 and NR3R4 = morpholine, piperidine, piperazine or pyrrolidine;
B = T or azetidinyl (all optionally substituted by 1-3 of T1);
Y = T or (thio)morpholinyl (all optionally substituted by 1-3 of T1);
T = phenyl, 3-10C cycloalkyl, 4-10C cycloalkenyl, pyrrolidinyl, piperidinyl, pyrrolyl, (iso)oxazolyl, (iso)thiazolyl, furanyl, thienyl, pyridyl, pyridazinyl, pyrimidinyl or pyrazinyl;
T1 = (1-20C)alkyl or alkoxy, amino, unsubstituted phenyl, azido, cyano, halo, 1-20C haloalkyl or nitro;
L = covalent bond, -C(=W)-, 1-20C alkylene, -NR5, -NR6, C(X)NR7-, 2-20C alkynylene, 2-20C alkenylene, -O-, S(O)t-, -NR6C(X)-, -C(X)NR6-, -NR6SO2NR7-, -NR6SO2-, -SO2NR6-, -OC(R1)(R2)- or -C(H)(R3)-;
R5 = R1 or aryl-1-20C-alkyl;
R6 and R7 = H, 1-20C alkyl or aryl-1-20C-alkyl;
R1 and R2 = R1;
R3 = OH or phenyl;
W = O, S or (=N-O-R6);
X = O or S; and
t = 0 - 2.

B and Y are attached through substitutable carbon atoms or nitrogen atoms in the ring. Provided that (I) is such that it excludes the combinations of:

(i) one of B or Y is phenyl or pyridyl and the other is phenyl or pyridyl;

(ii) B is phenyl, Y is cycloalkyl and L is -O-; and

(iii) R1 - R4 are H, B is cyclopropyl, Y is phenyl and L is a covalent bond.

ACTIVITY - Cytostatic; Anti-HIV; Antirheumatic; Antiarthritic; Anti-diabetic; Ophthalmological; **Immunosuppressive**; Antipsoriatic; Antiarteriosclerotic; Vasotropic; Vulnerary; Antiinflammatory; Dermatological; Antiulcer.

MECHANISM OF ACTION - Angiogenesis inhibitor; Ovulation inhibitor.

An endothelial cell migration assay was performed as given in Polverini, P. J. Et al., **Methods** Enzymol, 198:440-445 (1991). Human Microvascular Endothelial Cells (HMVEC) were used in the test. The inhibitory potencies against bFGF Induced Human Microvascular Endothelia Cell Migration of 6-(4'-nitro(1,1'-biphenyl)-4-yl)-1,3,5-triazine-2,4-diamine at 600 nm were 100%, compared to the positive control having % inhibition of 53%.

USE - As angiogenesis inhibitor, in the treatment of both primary and metastatic solid tumors and carcinomas of the breast; colon; rectum; lung; oropharynx; hypopharynx; esophagus; stomach; pancreas; liver; gall bladder; bile ducts; small intestine; urinary tract; female genital tract; endocrine gland; skin including hemoangiomas, melanomas, sarcomas arising from bone or soft tissues and Kaposi's sarcoma; tumors of the brain, nerves, eyes and meninges including astrocytomas, gliomas, glioblastomas, retinoblastomas, neuromas, neuroblastomas, schwannomas and meningiomass; solid tumors arising from hematopoietic malignancies such as leukemias and including chloromas, plasmacytomas, plaques and tumors of mycosis, fungoides and cutaneous **T-cell** lymphoma/leukemia, lymphomas including both Hodgkin's and non-Hodgkin's lymphomas; prophylaxis of **autoimmune diseases** including rheumatoid, immune and degenerative arthritis; ocular **diseases** including diabetic retinopathy, retinopathy of pre maturity, corneal graft rejection, retrolental fibroplasia, neovascular glaucoma, rubeosis, retinal neovascularization due to macular degeneration and hypoxia; abnormal neovascularization conditions of the eye; skin **diseases** including psoriasis; blood vessel **diseases** including hemagiomas and capillary proliferation within atherosclerotic plaques; Osler-Webber Syndrome; myocardial angiogenesis; plaque neovascularization; telangiectasia; hemophiliac joints; angiofibroma; wound granulation; **diseases** characterized by excessive or abnormal stimulation of endothelial cells including intestinal adhesions, Crohn's **disease**, atherosclerosis, scleroderma and hypertropic scars (i.e. keloids) and **diseases** which have angiogenesis as a pathologic consequence including **cat** scratch **disease** (Rochele minalia quintosa) and ulcers (Helicobacter pylori).

ADVANTAGE - (I) is a potent inhibitor of angiogenesis, having no severe systemic toxicity in humans and is safe for human use.

Dwg.0/0

L23 ANSWER 22 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2001-015913 [02] WPIDS
DOC. NO. CPI: C2001-004340
TITLE: Novel AW1791i, BG2211i, K1391i, K5111i and N1541i secreted proteins, their fragments and polynucleotides, useful in diagnostic and research assays and for treating, e.g. immune and proliferative **disorders** and for regulating hematopoiesis.

DERWENT CLASS: B04
INVENTOR(S): COLLINS-RACIE, L A; EVANS, C; JACOBS, K; LAVALLIE, E R; MCCOY, J M; MERBERG, D
PATENT ASSIGNEE(S): (GEMY) GENETICS INST INC
COUNTRY COUNT: 93
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000066148	A1	20001109	(200102)*	EN	67
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2000048136	A	20001117	(200111)		
EP 1198241	A1	20020424	(200235)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI					
JP 2002542799	W	20021217	(200312)		76

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000066148	A1	WO 2000-US11818	20000429
AU 2000048136	A	AU 2000-48136	20000429
EP 1198241	A1	EP 2000-930284	20000429
		WO 2000-US11818	20000429
JP 2002542799	W	JP 2000-615032	20000429
		WO 2000-US11818	20000429

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000048136	A	Based on	WO 2000066148
EP 1198241	A1	Based on	WO 2000066148
JP 2002542799	W	Based on	WO 2000066148

PRIORITY APPLN. INFO: US 1999-131596P 19990429
AN 2001-015913 [02] WPIDS
AB WO 200066148 A UPAB: 20011129
NOVELTY - Isolated human AW1791i, BG2211i, K1391i, K5111i and N1541i secreted proteins comprising sequences of 32, 67, 308, 160 or 120 amino acids (aa), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) isolated protein (P1) encoded by:

(a) polynucleotide which comprises the nucleotide sequence (I) of 990 base pairs (bp);

(b) polynucleotide comprising nucleotides 156-251 of (I);

(c) polynucleotide comprising the nucleotide sequence of the full length protein coding sequence of clone AW179-1i (ATCC 207186);

(d) polynucleotide encoding the full length protein encoded by the cDNA insert of clone AW179-1i (ATCC 207186);

(e) polynucleotide encoding a protein comprising amino acid (aa) sequence (II) of 32 aa; or

(f) polynucleotide encoding a protein comprising a fragment of 8

contiguous aa of (II);
(2) isolated protein (P2) encoded by:
(a) polynucleotide which comprises the nucleotide sequence (III) of 429 bp;
(b) polynucleotide comprising nucleotides 1039-1239 or 1090-1239 of (III);
(c) polynucleotide comprising the nucleotide sequence of the full length or mature protein coding sequence of clone BG221-1i (ATCC 207186);
(d) polynucleotide encoding the full length or mature protein encoded by the cDNA insert of clone BG221-1i (ATCC 207186);
(e) polynucleotide encoding a protein comprising amino acid (aa) sequence (IV) of 67 aa; or
(f) polynucleotide encoding a protein comprising a fragment of 8 contiguous aa of (IV);
(3) isolated protein (P3) encoded by:
(a) polynucleotide which comprises the nucleotide sequence (V) of 1606 bp;
(b) polynucleotide comprising nucleotides 49-972 of (V);
(c) polynucleotide comprising the nucleotide sequence of the full length protein coding sequence of clone K139-1i (ATCC 207186);
(d) polynucleotide encoding the full length protein encoded by the cDNA insert of clone K139-1i (ATCC 207186);
(e) polynucleotide encoding a protein comprising amino acid (aa) sequence (VI) of 308 aa; or
(f) polynucleotide encoding a protein comprising a fragment of 8 contiguous aa of (VI);
(4) isolated protein (P4) encoded by:
(a) polynucleotide which comprises the nucleotide sequence (VII) of 1869 bp;
(b) polynucleotide comprising nucleotides 90-569 of (VII);
(c) polynucleotide comprising the nucleotide sequence of the full length protein coding sequence of clone K511-1i (ATCC 207186);
(d) polynucleotide encoding the full length protein encoded by the cDNA insert of clone K511-1i (ATCC 207186);
(e) polynucleotide encoding a protein comprising amino acid (aa) sequence (VIII) of 160 aa; or
(f) polynucleotide encoding a protein comprising a fragment of 8 contiguous aa of (VIII); and
(5) isolated protein (P5) encoded by:
(a) polynucleotide which comprises the nucleotide sequence (IX) of 1448 bp;
(b) polynucleotide comprising nucleotides 125-505 of (IX);
(c) polynucleotide comprising the nucleotide sequence of the full length protein coding sequence of clone N154-1i (ATCC 207186);
(d) polynucleotide encoding the full length protein encoded by the cDNA insert of clone N154-1i (ATCC 207186);
(e) polynucleotide encoding a protein comprising amino acid (aa) sequence (X) of 127 aa; or
(f) polynucleotide encoding a protein comprising a fragment of 8 contiguous aa of (X).

All sequences are given in the specification.

ACTIVITY - Immunomodulator; antiinflammatory; immunosuppressive; cytostatic; vulnerary; osteopathic; antiarthritic; neuroprotective; antirheumatic; antiparkinsonian; nootropic; antiulcer; vasotropic; cerebroprotective; contraceptive; hemostatic; thrombolytic; antibacterial.

No biological data is given.

MECHANISM OF ACTION - None given.

USE - The proteins can be used in assays to determine biological activity and as markers for tissues in which the corresponding protein is preferentially expressed (either constitutively or at a particular stage

of tissue differentiation or development or in a disease state).

The proteins can be used as nutritional sources or supplements and may exhibit cytokine, cell proliferation or cell differentiation activity and may induce production of other cytokines.

The protein may exhibit immunomodulatory activity and can be useful in treatment of immune deficiencies and disorders. These immune deficiencies may be genetic, be caused by infections or may result from autoimmune disorders e.g. multiple sclerosis, rheumatoid arthritis and graft-versus-host disease.

The protein may be useful in regulation of hematopoiesis and in treatment of myeloid or lymphoid cell deficiencies and in treating various anemias or for use in conjunction with irradiation/chemotherapy to stimulate the production of erythroid precursors and/or erythroid cells. They are useful for supporting growth and proliferation of megakaryocytes and consequently of platelets, and used for prevention or treatment of various platelet disorders such as thrombocytopenia and/or in supporting the growth and proliferation of hematopoietic stem cells.

The protein may be used for bone, cartilage, tendon, ligament and/or nerve tissue growth or regeneration, as well as for wound healing and tissue repair and replacement and in the treatment of burns, incisions and ulcers. The protein may be used in the treatment of periodontal disease, and in other tooth repair processes and in the treatment of osteoporosis or osteoarthritis. They are also useful in tendon/ligament formation.

The protein may be useful for proliferation of neural cells and for regeneration of nerve and brain tissue, i.e. for the treatment of central and peripheral nervous system diseases and neuropathies, as well as mechanical and traumatic disorders, which involve degeneration, death or trauma to neural cells or nerve tissue and so is used in the treatment of diseases such as Alzheimer's disease, Parkinson's disease, Huntington's disease, amyotropic lateral sclerosis, and head trauma and cerebrovascular diseases such as stroke.

The proteins exhibit activin- or inhibin-related activities and are useful as a contraceptive to decrease fertility in female mammals and decrease spermatogenesis in male mammals or in advancement of the onset of fertility in sexually immature mammals, so as to increase the lifetime reproductive performance of domestic animals such as cows, sheep and pigs.

The proteins exhibit anti-inflammatory activity and can be used to treat inflammatory conditions such as septic shock, sepsis or systemic inflammatory response syndrome (SIRS).

Dwg.0/1

L23 ANSWER 23 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2000-452387 [39] WPIDS
DOC. NO. CPI: C2000-137936
TITLE: Inducing **T cell** receptor gene rearrangement for treating **autoimmune diseases** comprises contacting **T cells** with a CD40-**binding** agent.
DERWENT CLASS: B04 D16
INVENTOR(S): NEWELL, E; NEWELL, M K; WAGNER, D
PATENT ASSIGNEE(S): (UYVE-N) UNIV VERMONT & STATE AGRIC COLLEGE
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000039283	A1	20000706	(200039)*	EN	50

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ TZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES

FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS
LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL
TJ TM TR TT TZ UA UG UZ VN YU ZA ZW
AU 2000022157 A 20000731 (200050)
EP 1141240 A1 20011010 (200167) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
JP 2002533118 W 20021008 (200281) 66

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000039283	A1	WO 1999-US30930	19991222
AU 2000022157	A	AU 2000-22157	19991222
EP 1141240	A1	EP 1999-966655	19991222
		WO 1999-US30930	19991222
JP 2002533118	W	WO 1999-US30930	19991222
		JP 2000-591175	19991222

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000022157	A	Based on	WO 2000039283
EP 1141240	A1	Based on	WO 2000039283
JP 2002533118	W	Based on	WO 2000039283

PRIORITY APPLN. INFO: US 1998-114106P 19981229
AN 2000-452387 [39] WPIDS
AB WO 200039283 A UPAB: 20010410
NOVELTY - Inducing **T cell** receptor gene rearrangement comprises contacting a **T cell** with a CD40-**binding** agent that **binds** CD40 to induce **T cell** receptor gene rearrangement in the **T cell**.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a **method** for promoting **T cell** maturation comprising contacting an immature **T cell** with a CD40-**binding** agent that **binds** CD40 to promote maturation of the immature **T cell**;

(2) a **method** for inhibiting **T cell** receptor rearrangement comprising contacting a **T cell** expressing CD40 with an agent that inhibits CD40-induced **T cell** receptor rearrangement;

(3) a **method** for inducing **T cell** reactivity towards an antigen comprising introducing an amount of **T cells** and an amount of antigen presenting cells into a culture vessel and co-culturing the cells in the presence of a CD40 **binding** agent that **binds** CD40 to induce **T cell** receptor gene rearrangement and at least one antigen under conditions which induce the formation of **T cells** with specificity for the at least one antigen;

(4) a **method** for inhibiting environmental stress-induced cell death of a **T cell** comprising contacting a **T cell** expressing CD40, under environmental stress otherwise sufficient to induce cell-death, with a CD40-**binding** agent that **binds** CD40 and induces **T cell** receptor gene rearrangement to inhibit death of the cell expressing CD40

which would otherwise result from the environmental stress; and
(5) a **method** for enhancing environmental stress-induced cell death of a **T cell** comprising contacting a **T cell** expressing CD40 with a CD40-**binding** agent that **binds** CD40 and induces **T cell** receptor gene rearrangement, subjecting the CD40-**binding** agent bound **T cell** to an environmental stress sufficient to induce cell death.
ACTIVITY - **Immunosuppressive**; antiinflammatory; antithyroid; antidiabetic; antirheumatic; anarthritic; antianemic; opthalmological; antipsoriatic; nephrotophic; hepatotropic; virucide; dermatological; cytostatic.
No biological data is given.
MECHANISM OF ACTION - None given.
USE - The **methods** are used for inducing **T cell** receptor gene rearrangement, promoting **T cell** maturation, inhibiting **T cell** receptor rearrangement, inhibiting environmental stress-induced cell death, altering the specificity of a **T cell** towards an antigen, inducing **T cell** reactivity towards an antigen or enhancing environmental stress-induced cell death (all claimed). **T cell** affinity maturation towards a specific antigen can be inhibited and in particular for a self-antigen in an **autoimmune disease** which includes rheumatoid arthritis, uveitis, insulin-dependent diabetes mellitus, hemolytic anemias, rheumatic fever, Crohn's **disease**, Guillain-Barre syndrome, psoriasis, thyroiditis, Grave's **disease**, myasthenia gravis, glomerulonephritis, autoimmune hepatitis or systemic lupus erythematosus.
Inducing environmental stress-induced **T cell** death is carried out in a cancerous **T cell** or a self-reactive **T cell** where the environmental stress is a **chemotherapeutic agent** (claimed).
Dwg.0/6

L23 ANSWER 24 OF 29 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:316784 BIOSIS
DOCUMENT NUMBER: PREV200100316784
TITLE: Epstein-Barr virus (EBV)-associated post-transplant lymphoproliferative **disorder** (PTLD) after high-dose **immunosuppressive** therapy (HDIT) and autologous CD34-selected stem cell transplantation (SCT) for severe **autoimmune diseases**.
AUTHOR(S): Nash, Richard A. [Reprint author]; Dansey, Roger; Storek, Jan [Reprint author]; Sullivan, Keith M.; Pavletic, Steven; McDonagh, Kevin T.; Kraft, George H.; Mayes, Maureen D.; Forman, Stephen J.; Holmberg, Leona A. [Reprint author]; McSweeney, Peter A.
CORPORATE SOURCE: Fred Hutchinson Cancer Research Center, Seattle, WA, USA
SOURCE: Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 406a. print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology. San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 4 Jul 2001
Last Updated on STN: 19 Feb 2002

AB HDIT and autologous CD34-selected SCT are currently being evaluated for the control of severe **autoimmune diseases**. Autologous peripheral blood stem cells were mobilized with G-CSF (16 mug/kg/day) and CD34-selected. HDIT consisted of total body irradiation (800 cGy), cyclophosphamide (120 mg/kg) and **horse** anti-thymocyte globulin (ATG) (15 mg/kg/day) on days -5, -3, -1, +1, +3, +5. If patients had significant reactions to **horse** ATG on the skin test or during the subsequent intravenous administration, rabbit ATG (Sangstat, 2.5 mg/kg/day, administered on the same days) was started. Thirty-eight patients (median age 41 (range 23-60) years) with either multiple sclerosis (MS) (n=20) or systemic sclerosis (SSc) (n=18) have been treated. The median follow-up has been 7 (range 2-37) months. Two patients (MS=1; SSc=1) developed aggressive EBV-PTLD and died on day +53 and +64, respectively. Multi-organ clonal **B cell** infiltrates which were EBV-positive by molecular studies or immunohistology were identified at both autopsies. Both patients had positive skin tests for **horse** ATG and had been converted to rabbit ATG from the first dose. The development of EBV-PTLD in the first case was preceded by a severe acyclovir-resistant herpes simplex virus infection of the mouth which was treated with foscarnet and, in the second case, by a CMV pneumonia and gastroenteritis. Two patients who had partial courses of rabbit ATG because of a reaction to the intravenous infusion of **horse** ATG and 34 patients treated with only **horse** ATG had no significant reactivation of herpetic infection or developed EBV-PTLD. Although the numbers are limited, the time course and similarity of the two cases of EBV-PTLD support a relationship with the rabbit ATG. Rabbit ATG at the doses prescribed in this study was more **immunosuppressive** than **horse** ATG since it was only in the patients who received rabbit ATG in whom **T cells** were absent at day 28 in the peripheral blood. The differences between **horse** and rabbit ATG are not yet clearly defined and should not be considered interchangeable without further study. Patients with sensitivity reactions to **horse** ATG will not receive alternative antibody therapy.

L23 ANSWER 25 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2000-072622 [06] WPIDS
CROSS REFERENCE: 2001-638470 [73]
DOC. NO. CPI: C2000-020802
TITLE: Novel polynucleotides used to develop products for treating e.g. immune **disorders**, blood **disorders, autoimmune disorders** , allergies, inflammation, hyperproliferative **disorders** or infections.
DERWENT CLASS: B04 D16
INVENTOR(S): EBNER, R; RUBEN, S M
PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC; (EBNE-I) EBNER R; (RUBE-I) RUBEN S M
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9961617	A1	19991202	(200006)*	EN	169

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW

AU 9942087 A 19991213 (200020)
EP 1082433 A1 20010314 (200116) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
MX 2000011729 A1 20010601 (200235)
JP 2002516103 W 20020604 (200239) 233
US 2003003545 A1 20030102 (200305)
US 2003092133 A1 20030515 (200335)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9961617	A1		WO 1999-US11644	19990527
AU 9942087	A		AU 1999-42087	19990527
EP 1082433	A1		EP 1999-925886	19990527
			WO 1999-US11644	19990527
MX 2000011729	A1		MX 2000-11729	20001128
JP 2002516103	W		WO 1999-US11644	19990527
			JP 2000-551001	19990527
US 2003003545	A1	Provisional	US 1998-87340P	19980529
		Provisional	US 1998-99805P	19980910
		Provisional	US 1999-131965P	19990430
			US 1999-320713	19990527
US 2003092133	A1	Provisional	US 1998-87340P	19980529
		Provisional	US 1998-99805P	19980910
		Provisional	US 1999-131965P	19990430
		Div ex	US 1999-320713	19990527
			US 2002-153770	20020524

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9942087	A	Based on	WO 9961617
EP 1082433	A1	Based on	WO 9961617
JP 2002516103	W	Based on	WO 9961617

PRIORITY APPLN. INFO: US 1999-131965P 19990430; US 1998-87340P 19980529; US 1998-99805P 19980910; US 1999-320713 19990527; US 2002-153770 20020524

AN 2000-072622 [06] WPIDS
CR 2001-638470 [73]
AB WO 9961617 A UPAB: 20030603
NOVELTY - New isolated human interleukin-21 (IL-21) and IL-22 polynucleotides (PNs) and polypeptides are disclosed.

DETAILED DESCRIPTION - A novel isolated nucleic acid molecule (NAM) comprises a PN having a nucleotide sequence (NS) at least 95% identical to a sequence selected from:

(1) a PN fragment having a fully defined 705, 1067 or 1642 base sequence, given in the specification or a PN fragment of the cDNA sequence in ATCC Number 209666 or 209655;

(2) a PN encoding a polypeptide fragment having a fully defined 87, 160 or 197 residue amino acid sequence given in the specification, or the cDNA sequence in ATCC Number 209666 or 209655;

(3) a PN encoding conserved polypeptide domain (I), (II), (III), or (IV) of sequence (II), (III) or (IV) or the cDNA sequence in ATCC Number 209666 or 209655;

(4) a PN encoding a polypeptide epitope of sequence (II), (III) or (IV) or the cDNA sequence in ATCC Number 209666 or 209655;

(5) a PN encoding a polypeptide of sequence (II), (III) or (IV) or

the cDNA sequence in ATCC Number 209666 or 209655 having biological activity;

(6) a PN which is a variant or an allelic variant of sequence (II), (III) or (IV);

(7) a PN which encodes a species homolog of the polypeptide whose amino acid sequence is shown in sequence (II), (III) or (IV);

(8) a PN capable of hybridized under stringent conditions to any of the PNs as in (1)-(7), where the PN does not hybridize under stringent conditions to a NAM having a NS of only A residues or of only T residues; and

(9) a PN which is complementary to any of (1)-(8).

INDEPENDENT CLAIMS are also included for the following:

(1) a recombinant vector comprising an isolated NAM as in (1);

(2) a **method** of making a recombinant host cell comprising an isolated NAM as in the novelty, (1) or (2);

(3) a recombinant host cell produced by a **method** as in (2);

(4) an isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from:

(a) a polypeptide fragment of sequence (II) or the encoded sequence included in ATCC Number 209666, optionally having biological activity;

(b) a polypeptide domain or epitope of sequence (II) or the encoded sequence included in ATCC Number 209666;

(c) a mature form of a secreted protein or a full length secreted protein;a variant, allelic variant, or species homolog of sequence (II);

(5) an isolated antibody that **binds** specifically to an isolated polypeptide as in (4);

(6) a recombinant host cell that expresses an isolated polypeptide as above;

(7) a gene corresponding to a cDNA sequence of sequence (II), (III) or (IV).

ACTIVITY - Immunestimulatory; anticoagulant; **immunosuppressant**; antiasthmatic; antiinflammatory; cytostatic; antiviral; antibacterial; fungicide; vulnery.

MECHANISM OF ACTION - The IL-21 and IL-22 proteins modulate IL-6 secretion from NIH-3T3 cells. IL-21 and IL-22 proteins modulate immune system cell proliferation and differentiation in a dose-dependent manner.

USE - The polypeptides can be used for preventing, treating or ameliorating a medical condition (claimed). IL-21 and IL-22 polypeptide or PNs may be useful in treating deficiencies or **disorders** of the immune system, by activating or inhibiting the proliferation, differentiation, or mobilization (chemotaxis) of immune cells, treating or detecting deficiencies or **disorders** of hematopoietic cells, to modulate hemostatic or thrombolytic activity, in treating or detecting **autoimmune disorders**, treating asthma (particularly allergic asthma) or other respiratory problems, to treat and/or prevent organ rejection or graft-versus-host **disease** (GVHD), to modulate inflammation (e.g. septic shock, sepsis, arthritis, nephritis, **cytokine** or chemokine induced lung injury, inflammatory bowel **disease**, Crohn's **disease**, or resulting from over production of **cytokines**), to treat or detect hyperproliferative **disorders**, including neoplasms in the abdomen, bone, breast, digestive system, liver, pancreas, peritoneum, endocrine glands, eye, head and neck, nervous (central and peripheral), lymphatic system, pelvic, skin, soft tissue, spleen, thoracic and urogenital, hypergammaglobulinemia, lymphoproliferative **disorders**, sarcoidosis, Waldenstron's macroglobulinemia), to treat or detect infectious agents, e.g. viruses (e.g. arthritis, bronchiollitis, encephalitis , eye infections, chronic fatigue syndrome, hepatitis, meningitis, AIDS, pneumonia, chickenpox, measles, mumps, parainfluenza, rabies, the common cold, polio, leukemia, rubella, sexually transmitted **diseases**, or skin **diseases**) bacterial or fungal agents

(e.g. bacteremia, endocarditis, eye infections, gingivitis, opportunistic infections, respiratory tract infections, Lyme **disease**, **cat**-scratch **disease**, paratyphoid fever, food poisoning, pneumonia, gonorrhea and sexually transmitted **diseases**, meningitis, tuberculosis, lupus, gangrene, tetanus, rheumatic fever, urinary tract infections, wound infections), parasitic agents (e.g. scabies, dysentery, liver **disease**, malaria, toxoplasmosis), to differentiate, proliferate and attract cells, leading to the regeneration of tissues (e.g. repair, replace or protect tissue in wounds, burns, incisions or ulcers, osteoporosis, osteocarthritis, periodontal **disease**, liver failure, surgery, cosmetic plastic surgery, reperfusion injury) to proliferate and differentiate nerve cells (e.g. spinal cord **disorders**, head trauma, cerebrovascular **disease** and stroke), localized neuropathies and central nervous system **diseases** (e.g. Alzheimer's **disease**, Parkinson's **disease**, Huntington's **disease**, amyotrophic lateral sclerosis, and Shy-Drager syndrome). IL-21 and IL-22 polypeptides or PNS may also increase or decrease the differentiation or proliferation of embryonic stem cells and hematopoietic lineage, may be used to modulate mammalian characteristics such as body height, weight, hair color, eye color, skin, percentage of adipose tissue, pigmentation, size, and shape, to modulate mammalian metabolism affecting catabolism, anabolism, processing, utilization and storage of energy, to change a mammal's mental state or physical state by influencing biorhythms, caricadic rhythms, cicadian rhythms, depression (including depressive **disorders**), tendency for violence, tolerance for pain, reproductive capabilities, hormonal or endocrine levels, appetite, libido, memory, stress, or other cognitive qualities, as a food additive or preservative, such as to increase or decrease storage capabilities, fat content, lipid, protein, carbohydrate, vitamins, minerals, cofactors or other nutritional components. The polypeptides can also be used to identify **binding** partners. Mutations in the PNs or the presence or amount of expression or activity of the polypeptides can be used for diagnosing a pathological condition or a susceptibility to a pathological condition (claimed). Dwg.0/9

L23 ANSWER 26 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-571822 [48] WPIDS
DOC. NO. CPI: C1999-166855
TITLE: New isolated B7 and CTLA4 nucleic acids, used to develop products for treating, e.g. autoimmune and atopic **diseases**.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): SELLINS, K S; SIM, G; YANG, S
PATENT ASSIGNEE(S): (HESK-N) HESKA CORP
COUNTRY COUNT: 86
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9947558	A2	19990923	(199948)*	EN	148
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZA ZW					
AU 9931076	A	19991011	(200008)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9947558	A2	WO 1999-US6187	19990319
AU 9931076	A	AU 1999-31076	19990319

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9931076	A	Based on	WO 9947558

PRIORITY APPLN. INFO: US 1998-62597 19980417; US 1998-78765P 19980319

AN 1999-571822 [48] WPIDS

AB WO 9947558 A UPAB: 19991122

NOVELTY - Isolated B7 and CTLA4 nucleic acid molecules from **dogs** and cats are new.

DETAILED DESCRIPTION - (A) A novel isolated nucleic acid molecule (NAM) is selected from:

(a) a NAM having a nucleic acid sequence (NAS) that is at least about 80% identical to a NAS selected from the 24 sequences given in the specification, e.g. a sequence of 2830 base pairs (bp) or their fragment having at least about 12 bp;

(b) a NAM consisting of a NAS selected from the 4 sequences given in the specification, e.g. a sequence of 594 bp, and

(c) a NAM having a NAS selected from 8 sequences given in the specification, e.g. a sequence of 1856 bp, or their fragment having at least about 12 bp.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated NAM selected from:

(a) an isolated NAM having a NAS encoding:

(i) a B7 protein selected from a protein having an amino acid (aa) sequence (AAS) that is at least 80% identical to an AAS of 304 aa;

(ii) a protein comprising an epitope of the protein of (i);

(iii) a protein having an AAS that is at least 60% identical to an AAS of 329 aa;

(iv) a protein comprising an epitope of the protein of (iii);

(v) a protein having an AAS that is at least 80% identical to an AAS of 235 aa;

(vi) a protein comprising an epitope of the protein of (v);

(vii) a protein having an AAS that is at least 60% identical to an AAS of 280 aa;

(viii) a protein comprising an epitope of the protein of (vii);

(ix) a protein having an AAS that is at least 60% identical to an AAS of 332 aa;

(x) a protein comprising an epitope of the protein of (ix);

(xi) a protein having an AAS that is at least 80% identical to an AAS of 119 aa;

(xii) a protein comprising an epitope of the protein of (xi);

(b) a NAM comprising a complement of any of the NAS's as in (a);

(c) a NAM having a NAS encoding a CTLA4 protein selected from a protein having an AAS that is at least 90% identical to an AAS selected from sequences of 223 and 223 aa, and a protein comprising an epitope of the protein having an AAS that is at least 90% identical to an AAS selected from the two 223 aa sequences, and

(d) a NAM comprising a complement of any of the NAS's as in (c), where the B7 protein elicits an immune response against a naturally-occurring B7 protein, and where the CTLA4 protein elicits an immune response against a naturally-occurring CTLA4 protein (all sequences

are given in the specification);
(2) an isolated NAM selected from a NAM that encodes a naturally-occurring soluble mammalian B7 protein and a NAM comprising a complement of the NAM that encodes the protein;
(3) an isolated protein selected from:
(a) an isolated protein comprising a B7 protein selected from:
(i) a protein having an AAS that is at least 80% identical to the AAS of (1ai);
(ii) a protein comprising an epitope of the protein as in (i);
(iii) a protein having an AAS at least 60% identical to an AAS of (1aiii);
(iv) a protein comprising an epitope of the protein as in (iii);
(v) a protein having an AAS that is at least 80% identical to an AAS of (1av);
(vi) a protein comprising an epitope of the protein as in (v);
(vii) a protein as in (1avii), and a protein comprising an epitope of this protein;
(viii) a protein as in (1ax), and a protein comprising an epitope of this protein;
(ix) a protein as in (1axii), and a protein comprising an epitope of this protein, and where the B7 protein elicits an immune response against a naturally-occurring B7 protein, and
(b) an isolated protein comprising a CTLA4 protein selected from a protein having an AAS that is at least 90% identical to an AAS selected from the two 232 aa sequences and a protein comprising an epitope of this protein, where the CTLA4 protein elicits an immune response against a naturally-occurring CTLA4 protein;
(4) an isolated naturally-occurring soluble mammalian B7 protein;
(5) a recombinant molecule comprising a NAM as in (A), (1) or (2), operatively linked to a transcription control sequence;
(6) a recombinant virus comprising a NAM as in (A), (1) or (2);
(7) a recombinant cell comprising a NAM as in (A), (1) or (2), and
(8) an isolated antibody that selectively binds to a protein as in (3) or (4).
USE - They can be used for preventing or treating diseases, e.g. autoimmune diseases, allergic reactions, infectious diseases, tumor development, graft rejection, inflammation, arthritic and atopic diseases such as atopic dermatitis. They can be used in mammals such humans, dogs, cats, cattle, sheep or pets. The products can also be used for detection, diagnosis and drug screening.
Dwg.0/0

L23 ANSWER 27 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-479098 [40] WPIDS
CROSS REFERENCE: 1997-558518 [51]
DOC. NO. NON-CPI: N1999-356694
DOC. NO. CPI: C1999-140978
TITLE: Extra-corporeal treatment of cells, used for obtaining cells for treating e.g. tumors, **autoimmune disease**, transplant rejection, graft versus host disease or infections.
DERWENT CLASS: B04 P34
INVENTOR(S): EDELSON, R L
PATENT ASSIGNEE(S): (UYYA) UNIV YALE; (EDEL-I) EDELSON R L
COUNTRY COUNT: 23
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9938380	A1	19990805	(199940)*	EN	37

RW: AT BE CH CY DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: AU CA JP US
AU 9924750 A 19990816 (200002)
EP 1054591 A1 20001129 (200063) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE
JP 2002501726 W 20020122 (200211) 35
US 2002098469 A1 20020725 (200254)

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9938380	A1		WO 1999-US1729	19990128
AU 9924750	A		AU 1999-24750	19990128
EP 1054591	A1		EP 1999-904333	19990128
			WO 1999-US1729	19990128
JP 2002501726	W		WO 1999-US1729	19990128
			JP 2000-529128	19990128
US 2002098469	A1	CIP of	US 1996-621109	19960322
		CIP of	WO 1997-US4285	19970318
			US 1998-14705	19980128

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9924750	A	Based on	WO 9938380
EP 1054591	A1	Based on	WO 9938380
JP 2002501726	W	Based on	WO 9938380

PRIORITY APPLN. INFO: US 1998-14705 19980128; US 1996-621109 19960322; WO 1997-US4285 19970318

AN 1999-479098 [40] WPIDS
CR 1997-558518 [51]
AB WO 9938380 A UPAB: 20020829
NOVELTY - A novel **method** to enhance the level of expression of major histocompatibility complex (MHC) molecules on the cell membrane of cells withdrawn from a subject, comprises treating the cells with a **photoactivatable agent** to allow the agent to enter the cells, photoactivating the agent contained within the cells, and incubating the cells for at least 8 hours.
ACTIVITY - Anticancer; anti-rejection; **immunosuppressive**.
MECHANISM OF ACTION - Increased expression of major histocompatibility complex (MHC) molecules on the cell membrane of cells.
USE - The **methods** can be used for treating disease-effector cells comprising major histocompatibility complex (MHC) molecules which **bind** a disease-associated antigen, e.g. viral antigens, bacterial antigens, transplant antigens or tumor specific antigens (claimed). The treated cells can be used for treating e.g. leukemia, lymphoma, **autoimmune disease**, graft versus host disease, transplanted tissue rejection or infections and for producing vaccines.
Dwg.0/5

L23 ANSWER 28 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 1999-430394 [36] WPIDS
CROSS REFERENCE: 1997-480153 [44]; 1999-590729 [50]; 2000-594264 [56]; 2002-556723 [59]; 2002-626537 [67]; 2003-810570 [76]
DOC. NO. NON-CPI: N1999-320416
DOC. NO. CPI: C1999-126860
TITLE: New isolated apoptosis inducing molecule II polypeptides.

DERWENT CLASS: B04 C07 D16 S03
INVENTOR(S): EBNER, R; RUBEN, S M; ULLRICH, S; YU, G
PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC; (EBNE-I) EBNER R; (RUBE-I) RUBEN S M; (ULLR-I) ULLRICH S; (YUGG-I) YU G; (ZHAI-I) ZHAI Y; (ZHAN-I) ZHANG
COUNTRY COUNT: 84
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9935262	A2	19990715	(199936)*	EN	164
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZW					
AU 9921063	A	19990726	(199952)		
AU 9929721	A	19990906	(200003)		
EP 1044270	A2	20001018	(200053)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2002500043	W	20020108	(200206)		237

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9935262	A2	WO 1999-US242	19990107
AU 9921063	A	AU 1999-21063	19990107
AU 9929721	A	AU 1999-29721	19990219
EP 1044270	A2	EP 1999-901341	19990107
		WO 1999-US242	19990107
JP 2002500043	W	WO 1999-US242	19990107
		JP 2000-527646	19990107

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9921063	A	Based on	WO 9935262
AU 9929721	A	Based on	WO 9942584
EP 1044270	A2	Based on	WO 9935262
JP 2002500043	W	Based on	WO 9935262

PRIORITY APPLN. INFO: US 1998-27287 19980220; US 1998-3886 19980107; US 1998-75409P 19980220
AN 1999-430394 [36] WPIDS
CR 1997-480153 [44]; 1999-590729 [50]; 2000-594264 [56]; 2002-556723 [59]; 2002-626537 [67]; 2003-810570 [76]
AB WO 9935262 A UPAB: 20031125
NOVELTY - Isolated apoptosis inducing molecule II (AIM II) polypeptides and nucleic acids, are new.

DETAILED DESCRIPTION - (A) A novel isolated polypeptide comprises a member selected from:

(a) an apoptosis inducing molecule (II) (AIM II) N-terminal deletion mutant which has the amino acid sequence shown in sequence (II) (240 amino acids in length), provided that the amino acid sequence has a deletion of at least the first N-terminal amino acid residue but not more than the first 114 N-terminal amino acid residues of sequence (II);

(b) a polypeptide having an amino acid sequence at least 95%

identical to an amino acid sequence identical to (a); and

(c) a polypeptide having an amino acid sequence identical to that of (a) except for at least one amino acid substitution.

INDEPENDENT CLAIMS are also included for the following:

(1) an isolated polynucleotide (PN) 1169 bp (sequence given in the specification), encoding a polypeptide as in (A);

(2) a vector, and its **method** of production;

(3) a recombinant host cell and its **method** of production comprising introducing a recombinant vector as in (3) into a host cell;

(4) an isolated nucleic acid molecule (NAM) comprising a nucleotide sequence (NS) at least 95% identical to a sequence selected from:

(a) a NS encoding amino acids from 1 to 240 or 2 to 240 of sequence (II);

(b) a NS encoding an amino acid sequence encoded by a cDNA clone contained in ATCC Number 97689 or 97483;

(c) a NS encoding an AIM II polypeptide transmembrane domain, polypeptide intracellular domain or polypeptide having extracellular and intracellular domains but lacking the transmembrane domain; and

(d) a NS complementary to any of the NSs above;

(5) an isolated NAM comprising a PN which encodes an amino acid sequence of an epitope-bearing portion of an AIM II polypeptide as in sequence (II);

(6) (8) an isolated NAM selected from:

(a) at least 20 contiguous nucleotides of sequence (I) (1169 nucleotides in length), provided that the isolated NAM is not sequence (XX) (503 nucleotides in length) or any subfragments;

(b) a NS complementary to a NS as in (a); and

(c) (c) a NAM at least 20 nucleotides in length that hybridizes under stringent hybridization conditions to a NAM having a NS shown in sequence (I);

(7) an isolated AIM II polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from:

(a) amino acids from 1 to 240 or 2 to 240 in sequence (II);

(b) an amino acid sequence encoded by a cDNA clone contained in ATCC 97689 or 97483;

(c) an amino acid sequence of an extracellular domain, transmembrane domain or intracellular domain of the AIM II polypeptide;

(d) an amino acid sequence of a soluble AIM II polypeptide having all or part of the extracellular and intracellular domain but lacking the transmembrane domain; and

(e) the amino acid sequence of an epitope-bearing portion of any one of the polypeptides above;

(8) an AIM II polypeptide selected from a polypeptide comprising amino acid residues from 13 to 20, 23 to 36, 69 to 79, 85 to 94, 167 to 178, 184 to 196 or 221 to 233 in sequence (II);

(9) a **method** for making a recombinant vector comprising inserting an isolated NAM as in (4) into a vector;

(10) a recombinant vector produced by a **method** as in (9);

(11) a **method** of making a recombinant host cell comprising introducing a recombinant vector as in (10) into a host cell; and

(12) a recombinant host cell produced by a method as in (11).

ACTIVITY - Antiallergic; antiinflammatory; immunomodulator; antidiabetic; antibacterial; immunosuppressive; neuroprotective; osteopathic; antirheumatic; antiarthritic; dermatological.

MECHANISM OF ACTION - The effects of AIM II transduction on tumor growth were evaluated in vivo. When MDA-MB-231 cells were inoculated into mammary fat pads, AIM II expression significantly inhibited tumor formation of MDA-MB-231 in nude mice, whereas the vector control MDA-MB-231/Neo cells showed no change in tumor growth as compared with that of the parental MDA-MB-231 cells. Similar tumor suppression in the

MDA-MB-231/AIM II cells was also demonstrated in SCID mice. A histological examination of the tumors from AIM II expressing MDA-MB-231 cells or those from parental or vector control cells was performed. Parental or vector control MDA-MB-231 cells formed a large solid tumor mass filled with predominantly tumor cells with little or no cellular infiltrates.

In contrast, there was extensive necrosis observed even in small residual tumors formed by the MDA-MB-231/AIM II cells in nude mice. Furthermore, in AIM II expressing tumors, there is a significant increase in number of infiltrating neutrophil cells. The average number of neutrophils per mm2 tumor size in wild type, Neo control, and AIM II transduced MDA-MB-231 tumors were 101 plus or minus 26, 77 plus or minus 16, and 226 plus or minus 38 respectively, based on the immunohistological staining using Gr-1 monoclonal antibody. The inhibitory effect of AIM II on tumor suppression was further validated in the syngeneic murine tumor model. Local expression of AIM II in MC-38 murine colon cancer cells resulted in complete suppression of tumor formation in 8 out of 10 C57BL/6 mice. Local production of AMI II also dramatically prolonged the survival of mice bearing MC-38 tumors.

USE - The AIM II polypeptides mediate apoptosis by stimulating clonal deletion of T-cells. They can be used to treat lymphoproliferative disease which results in lymphoadenopathy, to stimulate peripheral tolerance and cytotoxic T-cell mediated apoptosis. They can be used to stimulate peripheral tolerance, destroy some transformed cell lines, mediate cell activation and proliferation and are functionally linked as primary mediators of immune regulation and inflammatory response. They can be used to treat autoimmune disease e.g. systemic lupus erythematosis (SLE), immunoproliferative disease lymphadenopathy (IPL), angioimmunoproliferative lymphadenopathy (AIL), immunoblastive lymphadenopathy (IBL), diabetes, multiple sclerosis, allergies, graft versus host disease.

Antagonists to AIM II polypeptides may be used to treat cachexia which is a lipid clearing defect resulting from a systemic deficiency of lipoprotein lipase, which is believed to be suppressed by AIM II, to treat cerebral malaria in which AIM II may play a pathogenic role, to treat rheumatoid arthritis by inhibiting AIM II induced production of inflammatory cytokines, such as IL-1 in the synovial cells, to prevent graft-versus-host rejection by preventing the stimulation of the immune system in the presence of a graft, to inhibit bone resorption and therefore to treat and/or prevent osteoporosis. They can also be used as anti-inflammatory agents, to treat endotoxic shock, and prevent activation of the HIV virus. The products can also be used for detection, diagnosis and prognosis. They can be used in mammals e.g. monkeys, apes, cats, dogs, cows, pigs, horses, rabbits and humans.

Dwg.0/2

L23 ANSWER 29 OF 29 WPIDS COPYRIGHT 2003 THOMSON DERWENT on STN
ACCESSION NUMBER: 2000-038343 [03] WPIDS
DOC. NO. CPI: C2000-009734
TITLE: Use of **T cell** epitope peptides for, e.g. preventing allergies.
DERWENT CLASS: B04 D16
INVENTOR(S): CONTI-FINE, B M
PATENT ASSIGNEE(S): (MINU) UNIV MINNESOTA
COUNTRY COUNT: 85
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9930736	A2	19990624	(200003)*	EN	219

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL

OA PT SD SE SZ UG ZW
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD
GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV
MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT
UA UG US UZ VN YU ZW
AU 9931799 A 19990705 (200003)
EP 1037663 A2 20000927 (200048) EN
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9930736	A2	WO 1998-US26787	19981216
AU 9931799	A	AU 1999-31799	19981216
EP 1037663	A2	EP 1998-967008	19981216
		WO 1998-US26787	19981216

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9931799	A	Based on	WO 9930736
EP 1037663	A2	Based on	WO 9930736

PRIORITY APPLN. INFO: US 1997-991143 19971216
AN 2000-038343 [03] WPIDS
AB WO 9930736 A UPAB: 20000118
NOVELTY - Preventing or inhibiting an indication or **disease** associated with aberrant, pathogenic or undesirable antibody production, particularly autoimmune or allergic **diseases** comprises administering a **T cell** epitope peptide.

DETAILED DESCRIPTION - A novel **method** (A) of preventing or inhibiting a **disease** associated with undesirable antibody production which is specific for a particular antigen, comprises administering to a mammal at least one **T cell** epitope peptide or a variant. The sequence of the epitope peptide comprises an immunodominant **T cell** epitope sequence which is less than the sequence of the antigen. The antigen comprises the immunodominant **T cell** epitope sequence.

INDEPENDENT CLAIMS are also included for the following:

(1) a **method** for tolerizing a mammal to an antigen by administering at least one peptide comprising an immunodominant epitope sequence, which is a partial sequence of the immunodominant antigen epitope;

(2) identifying an optionally immunodominant epitope sequence useful to tolerize a mammal by:

(a) independently contacting mammalian **T cells** samples with different peptides (where the mammal is sensitized to an antigen comprising the peptide); and

(b) detecting or determining whether the **T cells** in one sample proliferate relative to other samples and relative to **T cells** which were not exposed to a peptide;

(3) contacting at least 2 samples of CD4+ cells obtained from at least 2 individual mammals of the same species and of different immune response loci genotype, with a preselected peptide, and determining the degree of proliferation;

(4) a **method** of suppressing an immune response to virus-specific proteins present in a recombinant viral vector using an epitope peptide comprising an immunodominant sequence from the virus;

(5) a tolerogen comprising at least one isolated and purified peptide having an immunodominant epitope sequence;

(6) a tolerogen as in (5) where the peptide comprises a universal CD4+ epitope sequence;

(7) inhibiting or treating an antibody-mediated **disease** in a mammal, comprising:

(a) administering at least one peptide comprising an immunodominant epitope sequence, which is a partial sequence of the immunodominant antigen epitope; and

(b) subjecting the mammal to plasmapheresis; and

(8) preparing a chimeric mammal with reduced muscle strength, comprising engrafting a genetically immunodeficient subject with human hematopoietic cells, having or at risk of, a **disease** characterized by reduced muscle strength.

ACTIVITY - Dermatological; **immunosuppressive**; antiinflammatory; hemostatic; antianemic; antiallergic; antiasthmatic; antithyroid; antidiabetic.

MECHANISM OF ACTION - Gene therapy; antibody inhibitors.

USE - The **methods** can be used to specifically tolerize or down regulate the priming or activity of antigen-specific **T cells** of a mammal. The **methods** can be used to prevent or inhibit an indication or **disease** associated with antibody production to an antigen such as an endogenous antigen, e.g. acetylcholine receptor, insulin, growth hormone, factor VIII or factor IX, or an exogenous antigen e.g. a fungal antigen, a plant antigen, an antigen of a domestic **cat** or an antigen of a mite (claimed). They can be used to treat **autoimmune diseases**, e.g. myasthenia gravis, systemic lupus erythematosus (SLE), Graves' **disease**, autoimmune hemolytic anemia, autoimmune thrombocytopenia, autoimmune asthma, cryoglobulinemia, thrombic thrombocytopenic purpura, primary biliary sclerosis, pernicious anemia or pemphigus. They can be used for treating allergic **diseases**, e.g. allergic rhinitis, allergic asthma, atopic dermatitis, allergic gastroentheropathy, anaphylaxis, urticaria or angiodema. The **methods** can also be used in gene therapy for treating a **disease** such as hemophilia or diabetes or an indication such as adenosine deamidase deficiency, growth hormone deficiency, insulin deficiency, factor IX deficiency or factor VIII deficiency (claimed). The **methods** can also be used for **drug** screening.

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